TO TEST THE REPRODUCIBILITY OF CHANGES INDUCED IN LIPID LIPOPROTEIN PROFILE AFTER HIGH CHOLESTEROL FAT DIET IN HEALTHY INDIVIDUALS

THESIS

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C E R T I F I C A T E

This is to certify that the work entitled "TO TEST THE REPRODUCIBILITY OF CHANGES INDUCED IN LIPID LIPOPROTEIN PROFILE AFTER HIGH CHOLESTEROL FAT DIET IN HEALTHY INDIVIDUALS", which is being submitted as a thesis for M.D.(Medicine) examination 1991 of Bundelkhand University by Dr. Ashok Kumar Suri, has been carried out in the department of Medicine, M.L.B. Medical College, Jhansi.

He has put in the necessary stay in the department as per university regulations.

Dated: 1-12-91

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A C K N O W L E D G E M E N T

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(Ashok Kumar Suri)

C O N T E N T S

INTRODUCTION

I N T R O D U C T I O N

Atherosclerosis, which is a type of arteriosclerosis affecting large arteries, has emerged as the major cause of morbidity and mortality on this planet. It is the number one killer of mankind both below and above the age of 65 years in the developed nations. In the developing nations, the same trend is coming up.

Atherosclerosis is the nett result of interaction of many factors called risk factors that is age, hypertension, cigarette smoking, hyperlipidemia etc.

Various studies, showed a positive direct relation of hyperlipidemia with precocious atherosclerosis. This link weakened as age advanced. Hyperlipidemia has been recognised not only as a controlable, but reversible risk factor, has become the target of various studies, with the aim of finding means of lowering it. In this context much work has been done and is being done to study the effect of dietary contents on serum lipoproteins.

Most of the studies have established that dietary cholesterol and fat have a definite relation to serum lipids.

John et al (1982) demonstrated that modification of diet has led to progression or regression of atherosclerosis. In experimental animals similar findingswere observed by Stamler and Epstein (1972) and Flamina Fidanza (1974).

Conner (1977) found that cholesterol of dietary origin is transformed into other lipoproteins class especifically low density lipoprotein and contributes in elevation of total plasma cholesterol.

Similarly different type of fatty acids behave differently. Saturated fatty acids have hypercholesterolemic effect (Ahrens et al, 1984 and Keys et al, 1984). While monosaturated fatty acids have least effect, polyunsaturated fatty acids may even decrease serum low density lipoprotein and hence serum total cholesterol, but then there are inter and intra individual variations as observed by Keys et al (1979). Some workers hold the opinion that serum cholesterol is independent of dietary cholesterol intake in human subjects.

The fasting cholesterol level has been used in calculating the individual risk factor for coronary artery disease.

It seems that importance of fasting cholesterol level has been over emphasized. This is evident from the fact that more than 40% of young patients of documented coronary artery disease do not reveal raised fasting cholesterol level (Gregory et al, 1983). Yet they have rampent atherogenesis vascular imvolvement. What then was the mechanism into action in such cases ?

Zilversmit (1973) tried to explain this in terms of post prandial phenomenon. He postulated that

transient postprandial rise of VLDL, chylomicron and formation of several species of unusual lipoproteins may cause repeated cholesterol deposition in arterial wall over the years while fasting serum cholesterol level may remain well within normal limit over same duration.

This postulation was further strengthened by the identification of LDL receptors by Joseph Gold Stein et al (1974) and its role in serum cholesterol metabolism. The disappearance of radio active cholesterol within 20 minutes of intravenous injection from the vascular compartment, reflects its high dynamic state with the tissue cholesterol. Perhaps this dynamic equilibrium is achieved by the presence of LDL receptors and possibly some other hormonal and/or neurogenic reflexes not yet recognised, which might be regulating these LDL receptors.

Inspite of the above findings, we should not forget that majority (60%) subjects of coronary artery disease and hence atherosclerosis, (coronary artery disease is the best clinically evident proof of atherosclerosis) have a high fasting serum cholesterol levels and hence the importance of fasting serum lipids and lipoproteins in genesis of atherosclerosis cannot be over looked.

While studying the fasting serum lipid and lipoprotein profile in healthy subjects and effect of high cholesterol breakfast for a prolonged period of 7 days,

it was found that there is enormous individual variability in responses (Arora et al, 1984; 1985).

The studies indicated that after prolonged high cholesterol breakfast for 7 days, young subjects showed predominant rise of STC in the form of HDL cholesterol, while old subjects rise of LDL cholesterol (Arora et al, 1984; 85). Some persons behaved differently and showed a fall in total serum cholesterol.

Kingsburg (1960) showed that individuals response to high cholesterol diet varies enormously but remains contant for an individual over a long period of time.

Later on Anathan C Cohen et al (1988) found that the response to dietary fat (40 gm, 80 gm, and 120 gm) was practically in the form of dietary cream, uniform in the form of raised serum triglycerides but this uniformity disappeared 8 hours after ingestion. The maximum rise was 2 hour after ingestion.

Beynen et al (1985) studied the effect of cessation of egg consumption on serum cholesterol. They concluded that part of the serum cholesterol effect in response to dietary cholesterol is individually determined and is stable for at least 6 years.

Study of Grunda and Vega (1982) also arrived at the same conclusion that there is great individual variation to dietary cholesterol and on its basis they

introduced the terms hypo and hyper responders depending on the response. In the above study of Grunda and Vega reproducibility was not tested to establish whether these so called 'hypo and hyper responders' showed the same effect or not after a period of 5-6 years.

Beynen (1985) studied the effect of dietary cholesterol on serum cholesterol and found that there is great individual variation. He also described hypo and hyper responders but found that these results were only partly reproducible after 6 years.

The present study was undertaken to find out the results obtained in the study of Arora et al (1984 and 1985) respectively are reproducible or not after a gap of 5-6 years.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

With the turn of this century, more specifically during the last 50 years, coronary heart disease has turned out to be the greatest killer of man kind, at least in the developed Nations. India too does not lags behind in this race. The incidence of CHD are increasing alarmingly (Mathew et al, 1960; Banerjee et al, 1960; Mathur and Sapru, 1963). Atherosclerosis is the single most important lesion causing coronary artery disease.

Atherosclerosis as defined by WHO is a "Variable combination of changes of the intima consisting of focal accumulation of lipids, complex carbohydrates, blood and its constituents, fibrous tissues and calcium deposit in bombined form" (WHO, 1968).

Atherosclerosis which is a type of arterio-sclerosis is essentially a degenerative disease, associated with advancing age, affecting leage arteries particularly coronaries and cerebrals. At birth there is no athero-sclerosis, but in majority of children aged 10 years atherosclerosis develops in the form of fatty streak, second stage of atherosclerosis is fibrous plaque and finally the so called advanced lesion are formed.

Altered levels of plasma lipoprotein has been recognised as one of the most important factor in the development of atherosclerotic lesions. Among the plasma

lipoprotein elevated levels of STC and LDL and favours the development of atherosclerosis while HDL has a protective role.

Of the risk factors influencing atherosclerosis age, sex and genetic factors are irreversible while smoking, obesity, hyperlipidemia and hypertension are reversible factors.

Numerous studies from the world over have established hyperlipidemia as an important risk factor in the genersis of atherosclerosis.

Efforts were put in, to find various means to lower serum lipoproteins. Diet is an important instrument in achieving this goal.

Many studies have been published, establishing the relation of diet to serum lipids. Short term and long term feeding of diet, rich in cholesterol gave variable responses. Whether these responses were maintained for a long period or could be reproduced after a gap of 5-6 years has emerged as a subject of great controversy.

In experimental animals like rabbit it has been observed that cholesterol feeding evoked atherosclerosis, (otherwise unknown in these animals) and withdrawal of cholesterol was associated with regression of lesion.

SERUM TOTAL CHOLESTEROL(STC)

Effect of diet on serum cholesterol was a controversial chapter. Ankelkeys and Anderson et al (1965) after a large study arrived at a conclusion that serum cholesterol level is essentially independent of the cholesterol intake over the whole range of natural human diets.

Later on it has been proved in many studies that serum cholesterol has definite relation to dietary cholesterol.

Feeding of cholesterol rich diet for 2-8 weeks, raised serum choleaterol in blood (Arora et al, 1986; Messinger et al, 1950; Conner et al, 1961; and Deborah Applebaum et al, 1979).

Further, it was found that the direct relation of dietary cholesterol to serum cholesterol, was much valid when a dietary cholesterol load of upto 600 mg cholesterol was given. For diet containing more than 600 mg cholesterol increase in serum cholesterol was found, but was not in proportion to increase in dietary cholesterol.

Later on it was found that the effect of dietary cholesterol is also dependent on the basal cholesterol level.

In adolescents, with initial cholesterol levels greater than 200 mg%, a 50% decrease in cholesterol intake led to an appreciable drop (15.6%) in serum cholesterol

levels. The effect was much more modest 8.3% in those with lower initial levels of serum cholesterol (Mc Grandey et al, 1972).

Another big survey of school going children was undertaken, and children were divided into (1) low serum cholesterol level(80-130 mg%), (2) intermediate(157-180 mg%), (3) high cholesterol (194-260 mg%) level. Their mean daily intake of energy that is sugar, fat, saturated fat and cholesterol was calculated and no positive correlation between these three groups could be established (Weitman et al, 1978).

In seven different studies summarised recently significantly weak correlation were noted between serum lipids and dietary P/S ratio.

Mellies Glueck (1983) in the same context observed that saturated fattyacids in diet has hypercholesterolemic effect on plasma lipid while polyunsaturated fatty acid decrease serum cholesterol and low density lipoprotein. Monosaturated fatty acids have no effect but then there are individual variation as observed by Key et al, 1978 and Ahrens et al, 1972).

Serum cholesterol is in high dynamic state with tissue cholesterol, this became evident when single injection of radio active serum cholesterol was given intravenously and cholesterol disappeared within 20 minutes from vascular compartment. Thus we see, that serum

cholesterol is dynamic , having within subject variation and subject to subject variations.

HYPO AND HYPER-RESPONDERS

Grundy and Vega worked out the differences between individuals in the response of serum cholesterol to dietary saturated fatty acids. They observed that certain individuals showed only small responses and were turned hypo responders while others developed high degree of hyper cholesterolemia (Hyper-responders to dietary fats).

But then the existence of hyporesponders and hyper-responders came to criticism on the grounds that the results of one trial cannot established the presence of hypo or hyper-responders. Secondly reproducibility was not studied in those subject; Thirdly the difference could be explained, at least partly, on the basis of inbetween person variation.

Katan and Beynen (1985) conducted a study to see the effect of cessation of dietary egg on serum cholesterol in 1976. From this study 8 (5 male and 3 female) persons were picked. Four were hypo-responders and 4 were hyper-responders. In 1982 they were studied again for effect of cessation of dietary egg on serum cholesterol, the findings were that persisted to remain as hyporesponders and two as hyper-responders while other showed eradic respons, thus it was only partly reproducible after 6 years.

Vega and Grundy (1984) postulated that hypo and hyper-responders do exist, and that the cholesterolemic response for dietary cholesterol is individually determined and stable for at least 6 years.

A trial of such a small number cannot be taken as granted and matter remain open for further studies. A similar trial is being conducted in our department by Prof. R.C. Arora and S.B. Agarwal et al.

REPRODUCIBILITY

Katan and Beynen (1985) studied 25 men and 19 women in 1976. Random samples were taken to find serum cholesterol level. All these subjects took on an average 2 eggs daily. Consumption of egg was stopped for 3 weeks and effect on serum cholesterol level seen by taking 2 to 4 samples and their mean was calculated. He concluded that there is an individual variations in response. Some subjects showed fall in serum cholesterol while others showed rise, while few did not show any response.

The same subjects were studied again in 1982 under similar conditions after abstenance from egg in diet for 3 months. In 2nd trial other sources of cholesterol were also restricted as mean egg consumption was 1.7 egg (total cholesterol withdrawal was 537 mg/day in 1976 and 566 mg/day in 1982).

Results indicated a statistically significant decrease in level of serum cholesterol in 1976. In 1982

also statistically significant fall in serum cholesterol following cessation of egg was there, but the fall in serum cholesterol was approximately twice than that in 1976.

The study also showed that the response per subject was only partly reproducible. Hevertheless there was a significant positive co-relation between the response in 1976 and 1982.

Beynen and Katan (1985) also conducted two controlled dietary trials on the same subjects and found that the response in each subject was only partly reproducible. Interval of study being 6 years .

He also concluded that a change in the nature of fat in the diet, can influence serum cholesterol concentration more markedly than a change in the amount of dietary cholesterol can.

The series of experiments performed between 1966 and 1972 by Vegroesa et al was reanalysed. In these studies 130 subjects (82 men and 48 women) participated in an average in 3-4 experiment. It was found that subject found to over responder under respond in initial studies did not consistently behaved similarly in subsequent studies. This showed that the results were poorly reproducible.

EFFECT OF FEEDING OF HIGH CHOLESTEROL FAT DIET

HDL are complex family of particles, carrying about 20% of plasma cholesterol. These lipoproteins and complexes are defined by floatation in the ultra centrifuge between density 1.063 and 1.21 gm/ml, by the presence of major protein constituents i.e. apolipoproteins A_1 and A_2 and by alpha migration on electrophoresis.

Three classes of HDL are separated on the basis of floatation rates between 0-355. HDL_2 have rates in excess of 3.5, HDL_1, minor in quantity, is found at density less than 1.063, and over laps into the distribution of low density lipoproteins. Recently a distinct sub type of HDL called HDLs has been identified by Mahley and colleagues. HDLc is found in plasma of cholesterol fed animals and to a much smaller extent in humans fed on high cholesterol and high fat dies. HDLc differs from other HDL subtypes by presence of apolipoprotein Eo. This property confers an affinity for the low density lipoproteins receptor (Mahley and Weisgraber, 1978).

HDL may contain upto 25% cholesterol, 4% triglycerides. The ratio of cholesterol to triglyceride may show wide fluctuation. Cholesterol content of HDL raised after high cholesterol diet while triglyceride content of HDL increased in hypertriglyceridemia (Mistry et al, 1977; Weiswerller et al, 1977).

The bulk of HDL mass appears to arise from the interaction of precursor particle called nascent HDL,

secreted by the liver and intestines with lipids and proteins released during the catabolism of triglyceride rich lipoproteins. Part of HDL also arises from uptake and transfer of free cholesterol from cell membrane.

FACTORS AFFECTING HDL LEVELS IN HUMANS

Constitutional Factor

The normal range is 30-90 mg%. Women have higher level of HDL at all ages after puberty, about 20% more (Harrison Book of Int. Med.) exogenous androgens administration lowers HDL level in men (Furman et al, 1967). No change in HDL concentration has been reported during pregnancy (Kinnunen et al, 1980).

Evidences of autosomal dominant inheritance of low HDL level has been reported with high prevalence of coronary artery disease (Verganic et al, 1981). High level of HDL occurs in black American population (Tyrlor et al, 1975).

HDL level in males are stable till puberty, when a slight decline occurs and then remain stable till age of 55-60 years. At this age there is an increase, and than plateau in older age. In females there is a small but gradual increase in serum HDL to 60 years after this no age effect is apparent.

OBESITY AND HDL CHOLESTEROL

HDL levels are high in teen subjects and comparatively high in obese (Wilson et al, 1972). Increase HDL

association with increase in VLDL and triglyceride (Wilson et al, 1972).

PHYSICAL ACTIVITY AND HDL CHOLESTEROL

Atheletes cross country runners, tennis players, soccer players are associated with high level of serum HDL. Reduction of adiposity is not associated with increase in HDL level (Wellman et al, 1980). Exercise programmes are associated with increased serum HDL level. This is also true for post myocardial infarction exercise programme (Harrison).

ALCOHOL AND HDL CHOLESTEROL

Small doses of alcohol is associated with high level of HDL and decreased incidence of IHD. The mechanism and cause of this is not known.

RELATIONSHIP OF IHD AND HDL CHOLESTEROL

High level of HDL are associated with low incidence of IHD. Independently low level of serum HDL cholesterol is not taken as important risk factor because the difference in normal HDL and HDL level to be included as risk factor is narrow, and laboratory error in finding HDL are great. Still HDL is a better predictor of CAD compared to serum cholesterol.

RELATIONSHIP OF DIET AND HDL CHOLESTEROL

High dietary intake of cholesterol in form of

egg yolk per day is associated with increase in apolipoprotein E containing HDL sub species in human (Mahley et al, 1978).

Tan et al (1974) showed that level of HDL and serum apolipoprotein A-1 increases following high cholesterol and saturated fat diet.

In the series of Katan and Baynen (1984) they found that cessation of egg consumption was associated with fall in serum HDL. In this study it was also established that HDL level was higher in hyper-responders. This could be because of mass index which was low in hyper-responders and low body weight is associated with high HDL levels.

Effect of high cholesterol fat diet on HDL has been studied but its reproducibility after age of 5-6 years has not been studied.

LOW DENSITY LIPOPROTEIN(LDL)

LDL is generated by the degradation and removal of triglycerides from very low density lipoproteins in the plasma. Their density is in the range of 1.019 to 1.063, and they contain apoprotein B. More than 75% of total cholesterol present in plasma is in the form of LDL.

Function of LDL is to supply cholesterol to a variety of extrahepatic parenchymal cells such as adrenal cortical cell, renal cells lymphocyte, muscle cells.

LDL receptors are present on hepatic cells

and above mentioned extrahepatic cell by which the transport of cholesterol is facilitated and then this cholesterol is used in synthesis of bile acids, steroid, sex hormones. In humans about 80% of LDL is removed in this way while 20% is degraded by scavenger cells.

DIET AND LOW DENSITY LIPOPROTEIN (LDL)

In most animals high fat and cholesterol diet is associated with increased level of LDL. In man the response varies. In those subjects whose serum cholesterol rises after high fat diet, in them LDL also rises.

Deborah applebaum et al (1979) demonstrated significant rise of LDL level in volunteers, after feeding 5 gm of egg yolk cholesterol/day for 30 days.

Baud et al (1981) demonstrated that there was significant fall in level of LDL in five colunteers 3 hours and 5 hours after taking butter diet. They attributed this fall due to defect in VLDL hydrolysis by serum lipases and due to metabolic blocking in liver or adipose tissues.

In addition to this, this has also been shown that diet induced LDL molecules have large molecular size than those on low fat cholesterol diet (Rudel et al, 1979). Clair and Leight (1978) have reported that the diet induced, large LDL are capable of stimulating cholesteryl esterification and accumulation in smooth muscle cells to

a greater extent than are normal LDL. Diet induced apoprotein fraction changes in LDL have also been reported (Mahley et al, 1977; Rudel et al, 1979).

CONCEPT OF LDL RECEPTOR IN CONTROL OF STC

It is now considered that LDL receptors play a pivotal role in regulating the level of serum cholesterol (Kita et al, 1982). In rabbits, rats and hamsters more than half of the total LDL receptors are located in the liver. However, the precise distribution of these receptors in man is unknown.

Hepatic LDL receptors are suppressed whenever the liver's content of cholesterol increase or its demand for cholesterol is reduces. Thus receptor suppression occurs when a high cholesterol diet is consumed (Hui et al, 1981) or when bile acids are infused (Angelin et al, 1983). Conversely LDL receptors increased when hepatic cholesterol synthesis is blocked by drugs compactin or (Goldstein et al, 1982 and Bilheimer et al, 1983). When bile acid binding resins are given(Shephered et al, 1980). Fasting has also been shown to suppress LDL receptor in rabbits (Goldstein, 1982). LDL receptors can be stimulated by thyroxine (Thompson, 1981) and by pharmacologic doses of estrogen (Winder, 1980).

All the changes in receptor activity alter the rate of uptake of LDL by the liver and cause reciprocal changes in plasma LDL levels. Whenever hepatic LDL receptors are suppressed, the plasma LDL level rises,

conversely, whenever, these receptors are induced, the plasma LDL levels fall. In familial hypercholesterolemia the basic defect is reduced number of LDL pool is removed from the plasma daily by the receptors, whereas in familial hypercholesterolemia heterozygotes it is about 15%. This receptor deficiency results in accumulation of LDL into the plasma, leading to raised level and premature atherosclerosis.

TRIGLYCERIDE AND VERY LOW DENSITY LIPOPROTEIN(VLDL)

The level of serum triglyceride rises considerably after fat ingestion. There is a definite positive relation in serum triglyceride to amount of fat ingested (Nikkila and Kontinnen, 1962; Denborrough, 1963).

Cohen, Noakes and Benade (1988) worked upon 12 medical students feeding 40, 80, 120 gm of fat in form of 100, 200, 300 gm cream. Fasting triglyceride and cholesterol were taken.post prandial effect were seen at 2, 4, 6 hour interval. He found that till 6 hour there was an increase in serum triglyceride in proportion to quantity of fat consumed after 7 hour figures could not be correlated.

REPRODUCIBILITY

In the work of Cohen, Noakes and Benade et al, in eight subjects test was repeated after 3 weeks for test meal i.e. 80 gm fat. He found mean intrasubject variation of 14% post prandial lipemia of 19%.

Clefsky et al (1974) described biphasic plasma triglyceride curve in response to high fat diet. An initial peak at 1-3 hour while 2nd at 4-7 hour. Ist rise due to chylomicron while 2nd due to VLDL.

Arora and Kushwaha et al (1987) found significant difference in responses of fat load in normal subject and patients of diabetes and IHD. Inspired by this a triglyceride tolerance test was proposed.

Test meal composition has also been shown to effect serum triglyceride level significantly. Glucose given 1 to 1½ hour before or even after test meal eliminate the rise in serum triglyceride.

Long term studies on the effect of dietary protein in lipid level indicate that low protein intake is associated with depression of serum lipids (Olson et al, 1957).

In 1957, Havel concluded that increment in concentration of triglyceride in the serum following ingestion of fat is entirely due to VLDL.

Extra production of VLDL and triglyceride is due to secondary abnormality like obesity, excess cholesterol and carbohydrate intake, diabetes mellitus and nephrotic syndrome.

VLDL REMNANT OR BETA VLDL

These are smaller particles than VLDL and contains more cholesterol. Both of these quantities make VLDL remnants more atherogenic.

VLDL remnants occur in human plasma for transient period in response to high fat cholesterol diet. They may cause repeated cholesterol deposition in cells in the arterial wall over years. The beta VLDL either chylomicron remnant or hepatic lipoprotein may represent the atherogenic particles postulated several years ago by Zilversmit (1973).

Beta VLDL may represent the most significant diet induced changes in lipoprotein predisposing to accelerated atherosclerosis.

EXERCISE AND TRIGLYCERIDE

Exercise reduces serum triglyceride and possible explanation for reduction is that working muscle directly use triglyceride for energy production.

CHYLOMICRON

They are the larger lipoprotein particles containing dietary triglyceride and cholesterol. Chylomicron remnant is chylomicron particle which has given its free fatty acid and monoglyceride (triglyceride part).

Triglycefide remnant goes to liver where it is taken up by chylomicron remnant receptor and metabolised.

Presence of chylomicron in fasting sample is always abnormal. Chylomicron present in postprandial blood sample can be separated by creaming in the cold.

ATHEROSCLEROSIS AND LIPID LIPOPROTEIN LEVEL

SERUM TOTAL CHOLESTEROL

At STC level of 220 mg/dl the incidence of CAD is nearly twice than at STC level of 180 mg/dl (Kannet et al, 1977). Similarly patients with CAD have higher serum cholesterol (Cohen et al, 1977) atleast this is true for more than 60% of IHD patients.

SERUM TRIGLYCERIDE

Albrink et al (1959), Mahley et al (1989) have shown that patients of CAD has higher serum triglyceride.

LDL CHOLESTEROL

LDL which constitutes about 75 percent of the total serum cholesterol is more specifically associated with CAD than is total cholesterol. It has been known for many years that the reduction of elevated LDL in other primate species is followed by regression of arterio-sclerotic lesions in coronary arteries in large vessel (St. Clair, 1983). We have now conclusive evidence in humans that reducing elevated LDL cholesterol will reduce the incidence of clinical events attributable to coronary arteriosclerosis (the lipid research clinics, coronary primary prevention trial results, 1984).

HDL CHOLESTEROL:

HDL level have an inverse relationship with coronary artery disease (Gordon et al, 1977). The ability of HDL cholesterol to predict the developing of coronary atherosclerosis has been estimated to be four times greater than LDL cholesterol and eight time greater than total cholesterol (Gordon et al., 1977). Each 10 mg/dl change in HDL cholesterol concentration is associated with 50% alteration in cardiovascular risk (Brensike et al, 1984).

Sub classes of HDL can be fractioned by Zonal ultra centrifugation and include HDL_2 and HDL_3. Among these subgroups HDL_2 appears to have the strongest inverse relationship with CAD and accounts for different levels of HDL between men and women (Gofman et al,1954). The possible mechanism by which HDL cholesterol decreases atherosclerosis include :

1. Reversal of cholesterol transport from the peripheral cells to the liver for removal from the body (Miller and Miller, 1975).
2. Inhibition of LDL cholesterol uptake by cells at the LDL receptor sites.

AIMS OF THE STUDY

The following were the aims of study.

1. To test the reproducibility of basal fasting serum lipid profile prior to test meal.
2. To test the reproducibility of fasting serum lipid profile after 7 days of HCFB.
3. To test the reproducibility of variations in serum lipid profile following HCFB.
4. To assess the diet induced lipid risk.

MATERIAL AND METHODS

MATERIAL AND METHODS

The case material for the present study consisted of healthy male individuals who were employees of medical college, Jhansi, their children, and mess servants. Only those subjects were taken, who were studied upon by Prof. R.C. Arora, Dr. Gulab Gupta et al, 1984 and Prof. R.C. Arora, Vinod Gupta et al, 1985.

Informed consent was taken from every case. Detailed clinical examination was carried out. Detailed investigations were done after which the subject was declared healthy. Detailed history of their dietary habit was noted. Any change in dietary habit during last 6 years was noted. Any change in their habit of smoking consuming alcohol was noted. History regarding illness in last 6 years was also noted.

In all 21 subjects were studied. Subjects were divided into 2 groups (Group A and B).

Group A consisted of 13 subjects. One subject dropped out, hence in all 12 subjects were studied their age ranged from 13-23 years.

Group B consisted of 8 subjects. One subject dropped out, hence 7 subjects were studied their age ranged from 45-55 years.

Design of Test

All the subjects were asked to have their dinner at 7 PM on the previous night and not to take

any thing except water till next morning. Basal fasting sample(I) were taken at about 8 AM in the recumbent posture without producing venous stasis(Koerselman et al, 1961). After this they were given the test breakfast in place of routine breakfast for 7 days.

On 8th day fasting sample (II) was taken in the same way after an overnight fast of 12-14 hours. From 8th to 14th day test breakfast was withdrawn and replaced by routine breakfast. On 15th day, third fasting sample(III) was taken in the same manner after an overnight fast of 12-14 hours.

Each time 4-5 ml of blood was collected. Serum was separated within 4-6 hours and was tested for the following :

1. Serum Total Cholesterol(STC)

It was estimated using the enzymatic kit supplied by Ranbaxy (CHOD PAP Method was employed).

2. Serum Triglyceride (STG)

Estimation of serum triglyceride was done by enzymatic kit supplied by Ranbaxy using G.P.O. PAP method.

3. Serum High Density Lipoprotein (HDL)

It was estimated by enzymatic kit supplied by Ranbaxy using PT PAP method.

4. Serum Very Low Density Lipoprotein(VLDL)

It was calculated by using the formula given by Friedwald et al (1972).

VLDL = STG/5 (This formula is valid till STG value is less than 400 mg%).

5. SERUM Low Density Lipoprotein(LDL)

It was estimated by using the formula given by Fredrickson et al (1972).

LDL = STC - (STG/5 + HDL) mg%.

PROTOCOL

Test diet(High cholesterol fat breakfast) was given to the subject from day 1st to 7th in place of routine breakfast. Test diet consisted of :-

- 4 slices of bread of average size.
- 25 gms of amul pasturized butter.
- 2 eggs omlette prepared in 20 gms vanaspati ghee.
- 250 ml of whole fat sweatened buffalo milk.
- 1 gm of crystalline cholesterol dissolved in milk.

The protocol and method was exactly the same as in the prior study of 1984-85.

Reproducibility of STC, STG and HDL was studied in both the groups and at individual level. Reproducibility of HCFB induced changes was studied . Diet induced risk was studied.

It was worked out on the basis of an arbitrary scale as follows :

GROUP A

Low risk = STC level less than 200 mg%.

LDL/HDL ratio less than 3.

Moderate risk = STC level more than 200 mg%.

LDL/HDL ratio less than 3 OR

OR

STC level less than 200 mg%

LDL/HDL ratio 3 or more than that.

High risk = STC level more than 200 mg%

LDL/HDL ratio 3 or more than 3.

GROUP B

In group B cut out point for STC level was taken 220 mg% instead of 200 mg%.

OBSERVATIONS

OBSERVATIONS

The present study was conducted on 21 subjects. All were males in age group of 13-55 years. These subjects were studied in 1984-85 also. The protocol and method of the study had remained same at the two occasions. In the present study the subjects were divided into two group A and group B.

GROUP 'A'

Table 1 : General characteristics of subjects of group A (N=12).

Characteristics	No.of cases	Percentage
OCCUPATION		
Student	6	50.00
Mess Servant	4	33.30
Auto driver	2	16.70
PHYSICAL ACTIVITY : Moderate	12	100.00
DIETARY HABIT		
Vegetarian taking egg	5	41.70
Non vegetarian	7	58.30
SMOKING/ALCOHOLISM		
Non smokers	12	100.00
Non alcoholics	12	100.00

Group A consisted of 13 cases (No. 1 to 13). Case No. 2 did not complete the study. Hence in all 12 subjects were studied. All the subjects were male

in age group of 13 to 23 years, with mean age of 18±2.86 years and mean weight of 49.75±7.18 kg. Their general characteristics are given in table 1.

GROUP B

It consisted of 8 subjects(No. 14 to 21). Case No. 21 withdrew from the study. Hence in all 7 subjects were studied. All the subjects were male in age group of 45 to 55 years with mean age of 48±5.7 years and mean weight of 58±3 kgs. Their general characteristics are given in the table 2.

Table 2 : General characteristics of subjects of group B (N=7).

Characteristics	No.of cases	Percentage
OCCUPATION		
Class IV employees(Ward/boy, Chowkidar, Sweeper).	7	100.00
PHYSICAL ACTIVITY		
Sedentary	-	-
Moderate	5	71.40
Heavy	2	28.60
DIETARY HABIT		
Vegetarian taking egg	4	57.40
Non vegetarian	3	42.60
SMOKING HAVIT		
Smokers 7 10 bidi/day	6	85.70
Occasionally	1	14.30
FAT CONSUMPTION		
Low fat intake	6	85.70
High fat intake	1	14.30
ALCOHOL CONSUMPTION		
Regular alcoholic	2	28.60
Non alcoholic or take occasionally	5	71.40

COMPARATIVE ACCOUNT OF STC LEVEL IN TWO STUDIES

GROUP 'A'

Table 3 : Comparative account of STC levels in two studies(Mean±S.D. mg/dl).

Year	STC		
	I	II	III
1985	156.75±39.30	190.25±36.15	165.25±31.10
1990	162.90±41.10	163.90±19.64	150.75±30.70
C.S.D.	39.45	31.46	31.00

Mean serum total cholesterol prior to test meal (I) was 156.75±39.40 mg/dl in 1985 and 162.90±41.1 mg/dl in 1990. The differences were statistically insignificant (p >0.05). That is to say that fasting serum cholesterol level with routine meals was reproducible after a gap of 5 years.

Following 7 days of HCFB, the serum total cholesterol (II) in 1985 was 190.25±36.15 mg/dl&in 1990 it was 163.90±19.64 mg/dl. The difference was statistically significant and hence it was observed that HCFB induced STC levels were not reproducible after a gap of 5 years.

After 7 days of cessation of HCFB STC (III) level in 1985 was 166.25±31.1 mg/dl and in 1990, it was 150.75±30.70 mg/dl. The difference was statistically significant and hence it was not reproducible.

GROUP 'B'

In group B the difference in fasting STC level prior to test meal in 1984 and 1990 was statistically

insignificant and thus was reproducible while fasting STC level after 7 days of HCFB (II) and 7 days after cessation of HCFB (III) was not reproducible as the differences observed in 1984 and 1990 were statistically significant.

TABLE 4 : Comparative account of STC level in two studies (Mean±S.D. mg/dl).

Year	STC		
	I	II	III
1984	159.14±27.35	201.57±52.54	179.57±17.87
1990	156.14±23.65	175.70±40.53	171.60±15.79
C.S.D.	27.00	47.00	16.70

This was observed that only fasting STC prior to test meal was reproducible in 1990 in both the groups.

COMPARATIVE ACCOUNT OF SERUM TRIGLYCERIDE LEVELS IN TWO STUDIES

GROUP 'A'

TABLE 5 : Comparative account of STG levels in two studies (Mean±S.D. mg/dl) (n=12).

Year	STG		
	I	II	III
1985	125.90±18.25	149.50±21.45	134.20±21.70
1990	119.90±39.20	129.60±34.14	131.90±29.54
C.S.D.	30.03	29.68	25.31

Fasting STC (I) levels was 125.90±18.25 mg/dl in 1985 and in 1990 it was 119.90±39.20 mg/dl. The difference was statistically insignificant and thus fasting STG (I) was reproducible.

Fasting STG after 7 days of HCFB (II) was not reproducible in 1990. The difference was statistically significant as is evident from the above table 5.

Seven days after cessation of HCFB the fasting STG (III) in 1985 was 134.20±21.7 mg/dl while in 1990 it was 131.90±29.54 mg/dl. The difference was statistically insignificant and hence it was reproducible.

GROUP 'B'

TABLE 6 : Comparative account of STG level in two studies (Mean±S.D. mg/dl)(N=7).

Year	STG		
	I	II	III
1984	94.14±22.80	106.50±23.30	138.30±45.20
1990	105.00±52.80	117.14±41.43	136.70±39.80
C.S.D.	14.76	33.38	40.90

In group B fasting STG (I) prior to test meal was 94.14±22.80 mg/dl in 1984. It was 105.0±52.8 mg/dl in 1990. The difference was statistically insignificant and hence reproducible.

After HCFB (II), the difference observed in 1984 and 1990 were statistically significant and hence non reproducible.

After 7 days of cessation of HCFB the STG (III) level in 1984 was 138.30±45.20 mg/dl and in 1990 it was

136.70±39.80 mg/dl. The difference was statistically insignificant and hence it was reproducible.

There was a difference in behaviour of group A and B in reproducibility of STG while STG II was not reproducible in either group. STG (III) was reproducible in both the groups.

STG(I) was reproducible in group A as well as in group B.

COMPARATIVE ACCOUNT OF HDL CHOLESTEROL LEVELS IN TWO STUDIES

GROUP 'A'

TABLE 7 : Comparative account of HDL levels in two studies (Mean±S.D. mg/dl)(N=12).

Year	HDL		
	I	II	III
1985	47.00±21.00	73.90±21.30	56.75±23.50
1990	42.00±10.70	52.30±14.90	45.90±13.30
C.S.D.	16.53	21.10	19.46

In group A the difference in HDL (I) in 1990 was statistically significant when compared to that of in 1985 and thus it was reproducible.

HDL level(II) and 7 days after cesaation of HCFB (III) the differences in their values in 1985 and 1990 were statistically significant and hence were not reproducible.

GROUP 'B'

TABLE 8 : Comparative account of HDL levels in two studies (Mean±S.D. mg/dl)(N=7).

Year	HDL		
	I	II	III
1984	65.60±7.32	76.85±25.00	61.00±4.04
1990	41.14±17.14	37.57±7.99	36.25±6.85
C.S.D.	17.90	27.12	13.94

In group B, fasting HDL prior to test meal was 65.60±7.32 mg/dl in 1984 and 41.14±17.14 mg/dl in 1990. The difference was statistically significant Hence it was not reproducible.

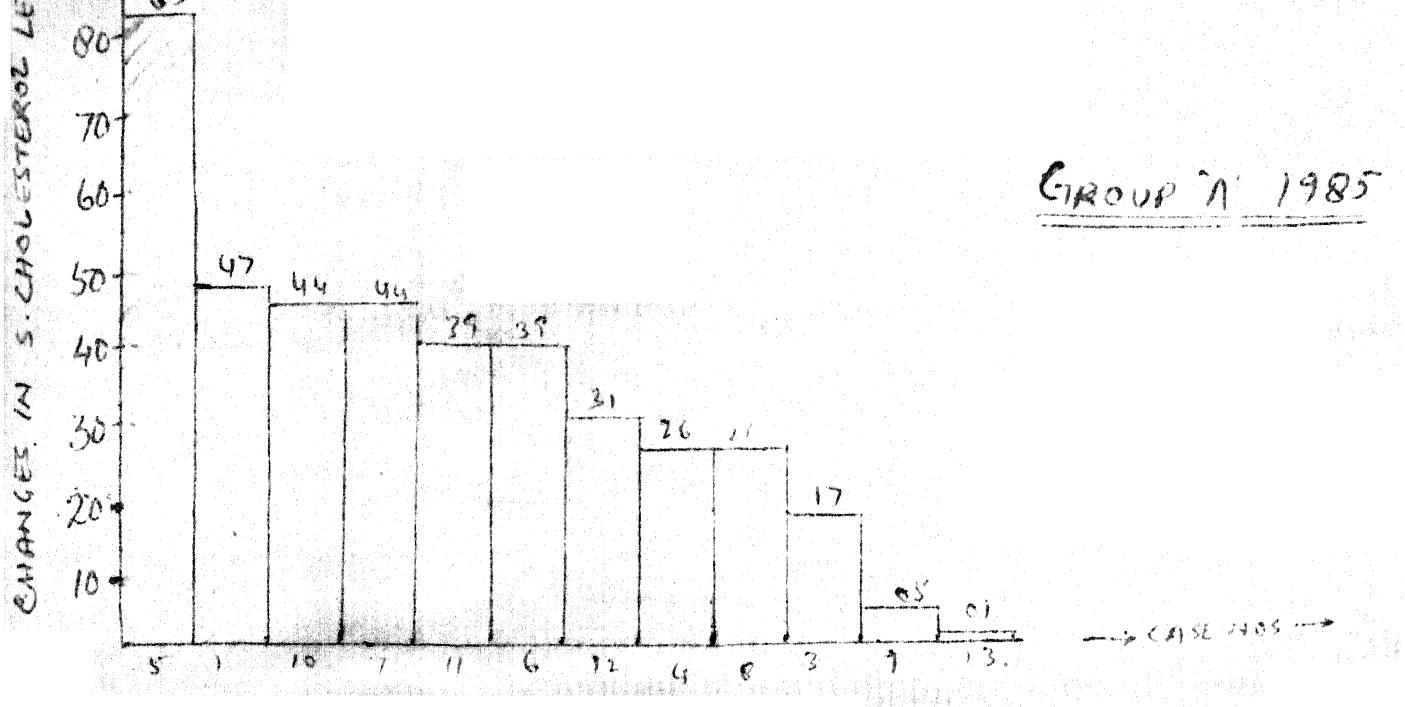
After 7 days of HCFB, the difference observed in 1984 and 1990 were statistically significant($p < 0.05$) and hence these were not reproducible.

After 7 days of cessation of HCFB, HDL(III) was showed a difference which was statistically significant and hence it was not reproducible.

HCFB INDUCED VARIATIONS IN SERUM LIPID PROFILES

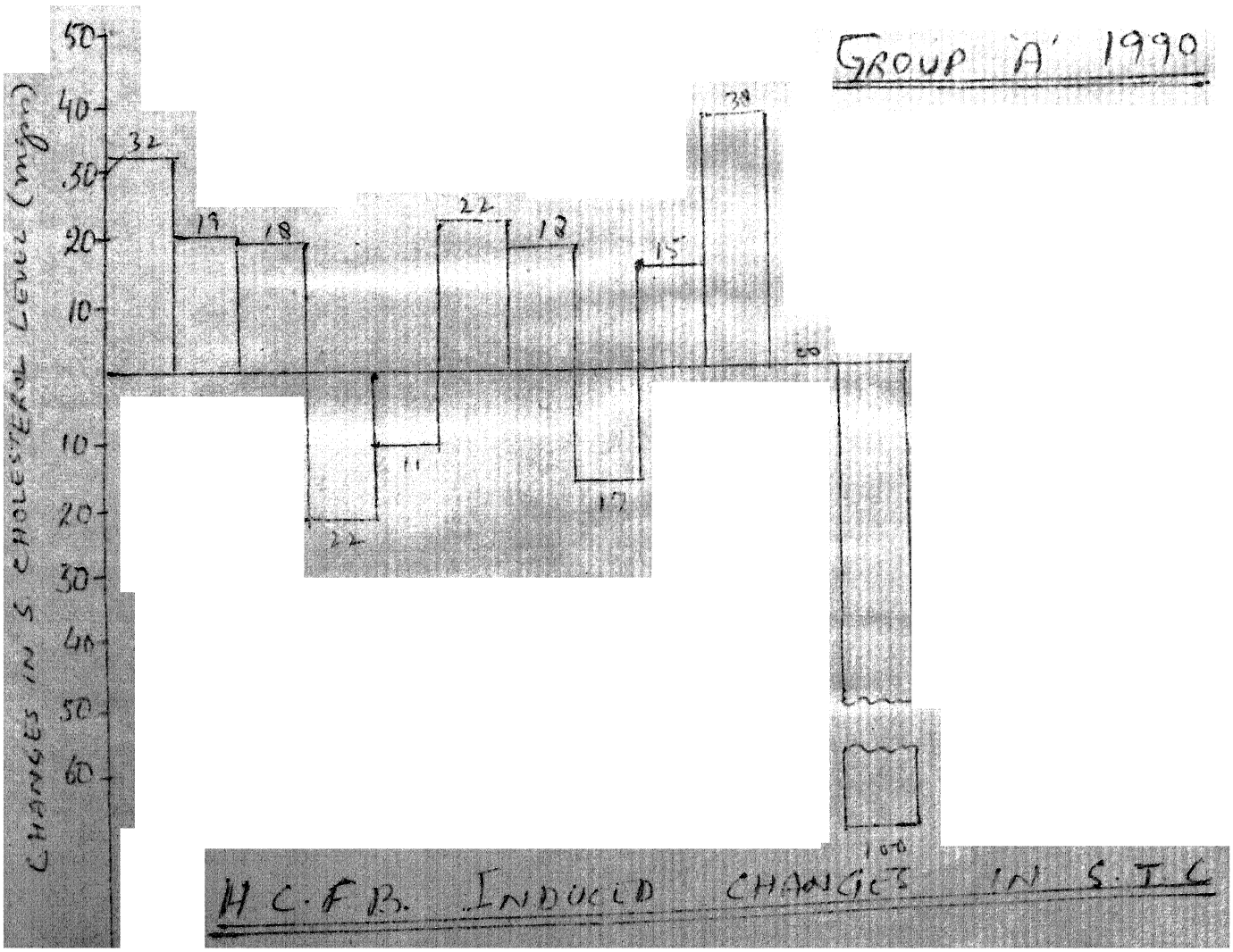
TABLE 9 : Comparative account of HCFB induced changes in serum lipid profile in group A (Mean±S.D. mg/dl)(N=12).

Year	STC	STG	HDL
1985	33.50±21.69	23.90±17.39	26.92±14.78
1990	26.00±25.17	17.17±14.53	12.00±10.95
C.S.D.	23.29	16.05	14.83



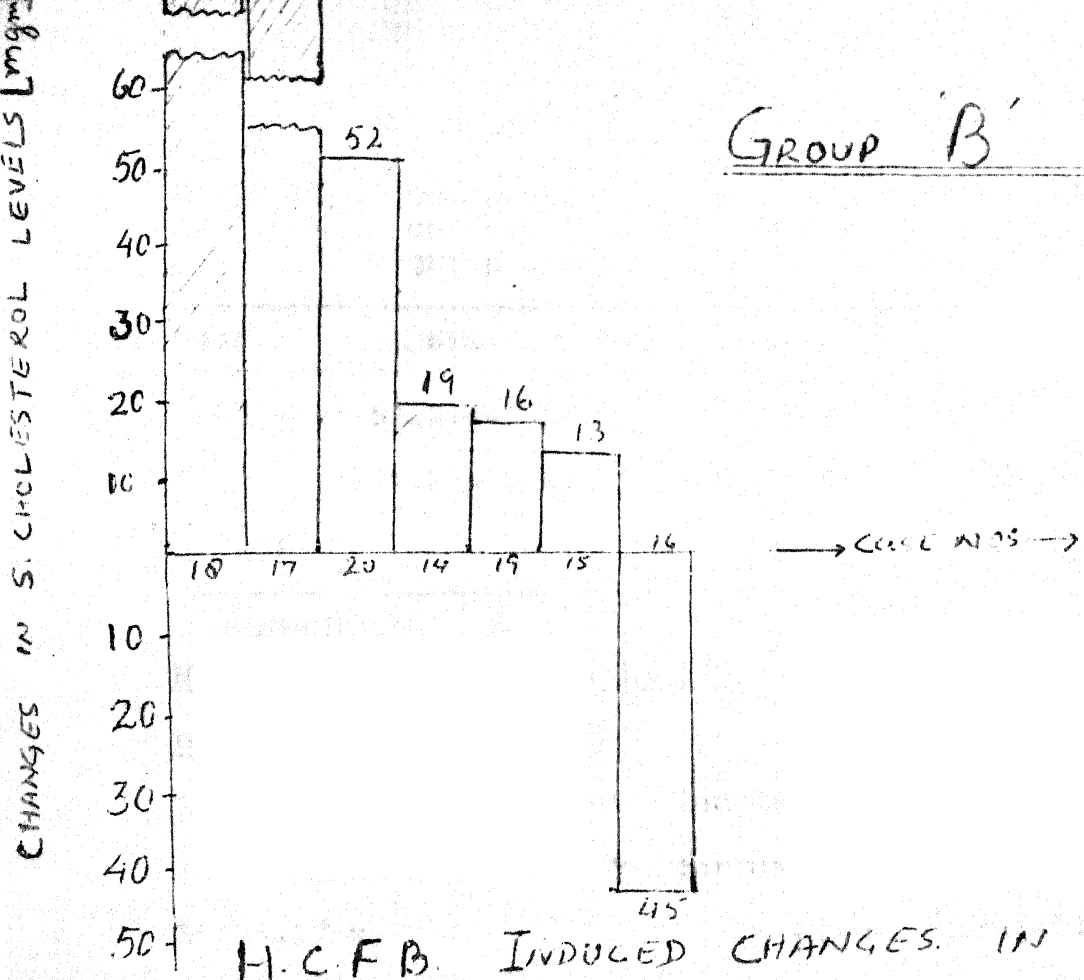
GROUP 'A' 1985

H.C.F.B. INDUCED CHANGES IN S.T.C.



GROUP 'A' 1990

H.C.F.B. INDUCED CHANGES IN S.T.C.


GROUP 'B' 1984
CHANGES IN S. CHOLESTEROL LEVELS [mgm]
60
50
40
30
20
10
52
19
16
13
10
17
20
14
19
15
16
10
20
30
40
50
41.5
H.C.F.B. INDUCED CHANGES IN S.T.C.

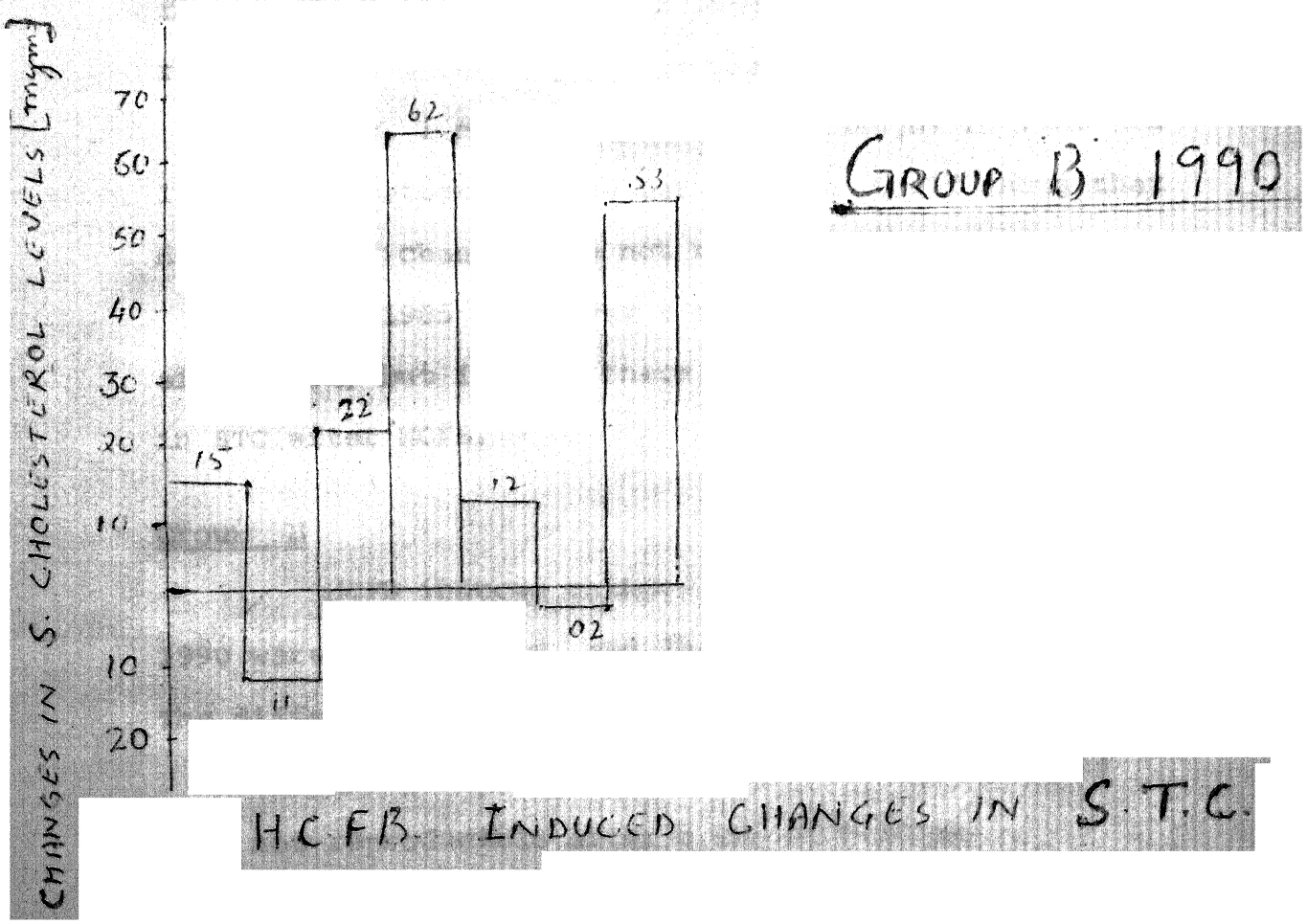

GROUP B 1990
CHANGES IN S. CHOLESTEROL LEVELS [mgm]
70
60
50
40
30
20
10
20
62
33
22
15
12
11
02
HCFB. INDUCED CHANGES IN S.T.C.

TABLE 10 : Comparative account of HCFB induced changes in serum lipid profile in group B (Mean ±S.D. mg/dl) (N=7).

Year	STC	STG	HDL
1984	52.29±42.59	23.71±15.66	20.71±20.75
1990	25.29±22.93	33.14±25.39	10.28±9.19
C.S.D.	35.72	20.85	16.35

HCFB Induced Variation in STG

Group A

The mean of HCFB induced changes was 33.50± 21.69 mg/dl in 1985. The corresponding mean in 1990 was 26±25.17 mg/dl. The difference was statistically significant ($P < 0.05$). Thus HCFB induced changes were not reproducible after a gap of 5 years in young age group.

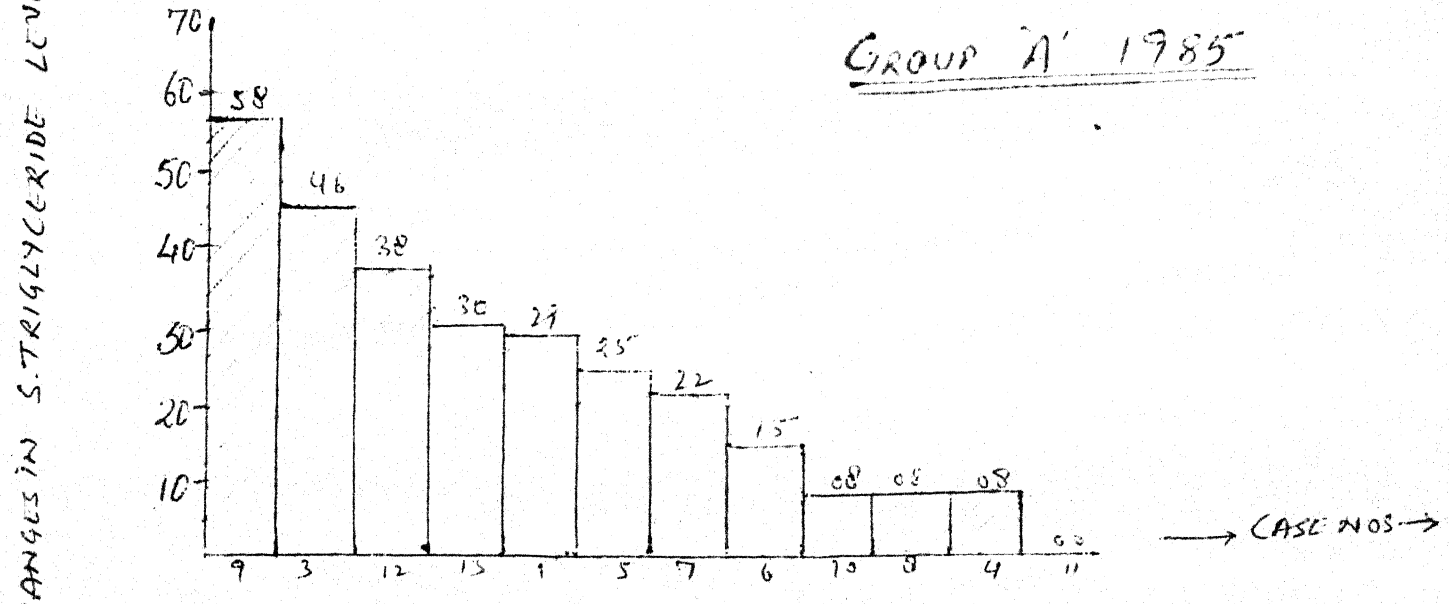
Fig. 1 shows HCFB induced changes in 1985 and 1990. The pattern of the bar diagram itself shows that HCFB induced changes are not reproducible.

In 1985 there was a significant rise in STC after HCFB, but in 1990 there was no significant rise in STC after HCFB.

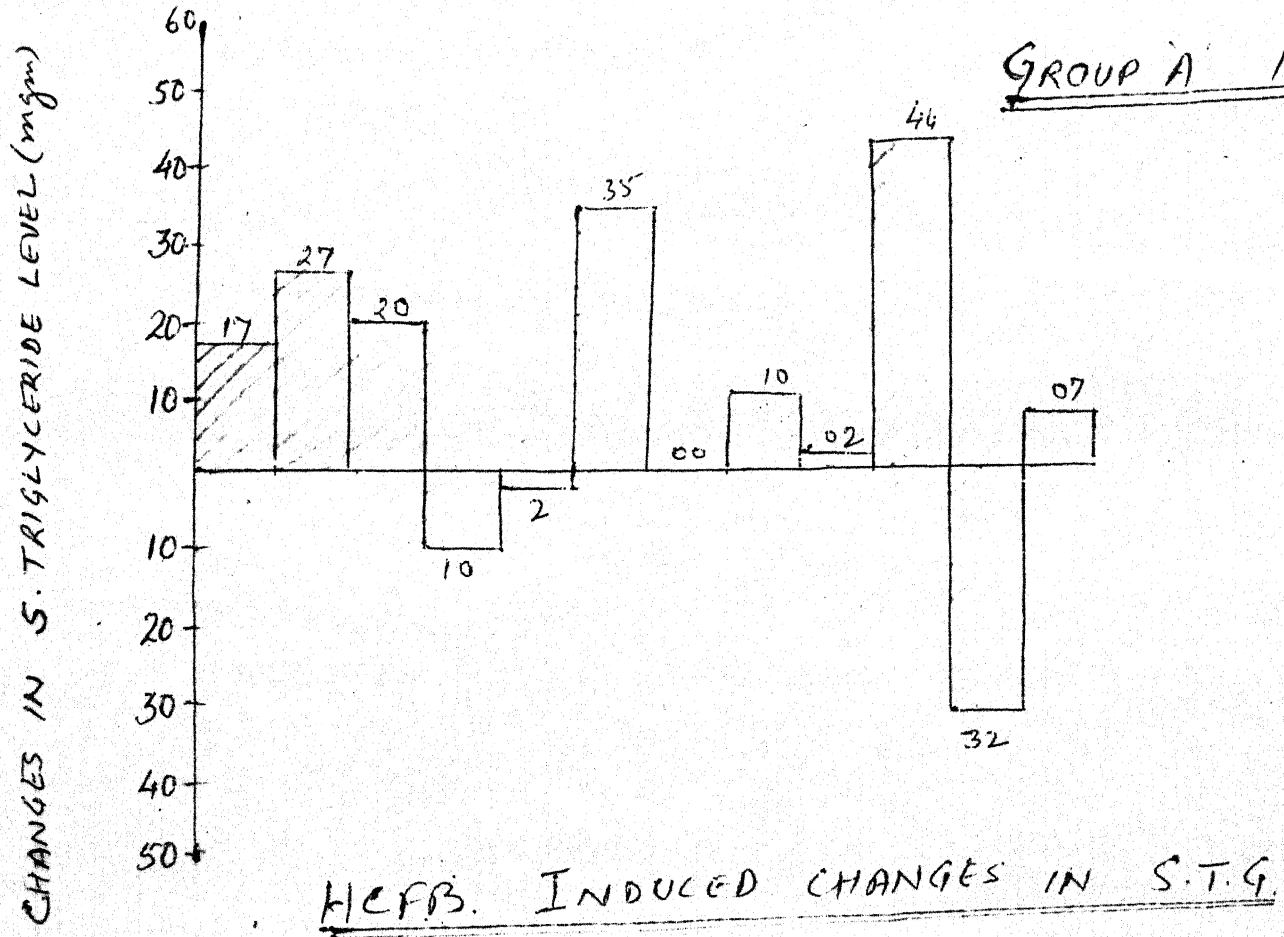
Group B

HCFB induced changes in STC level in 1984 and 1990 were 52.29±42.59 and 25.29±22.93 mg/dl respectively. The difference was statistically significant ($p < 0.05$).

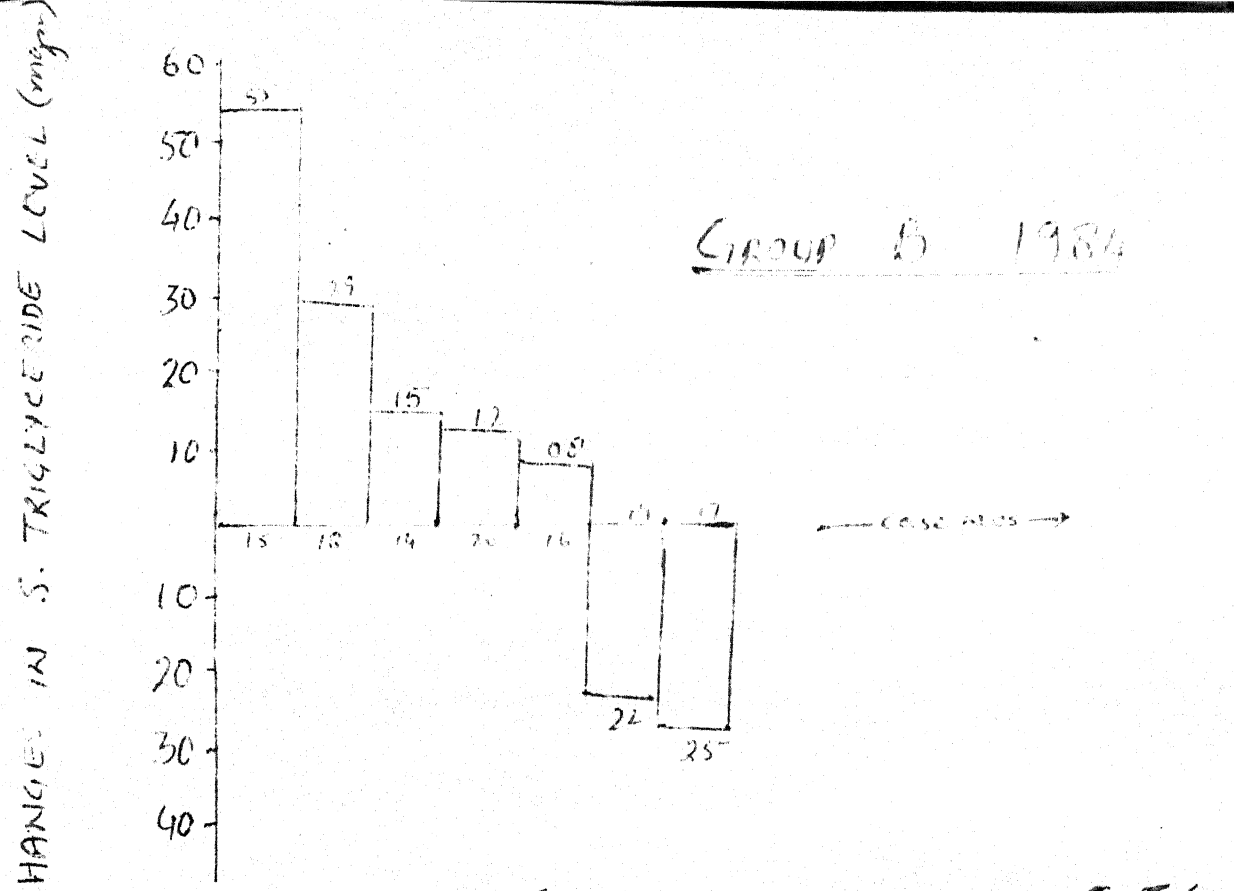
HCFB induced changes in middle aged persons was not reproducible after a gap of 6 years.



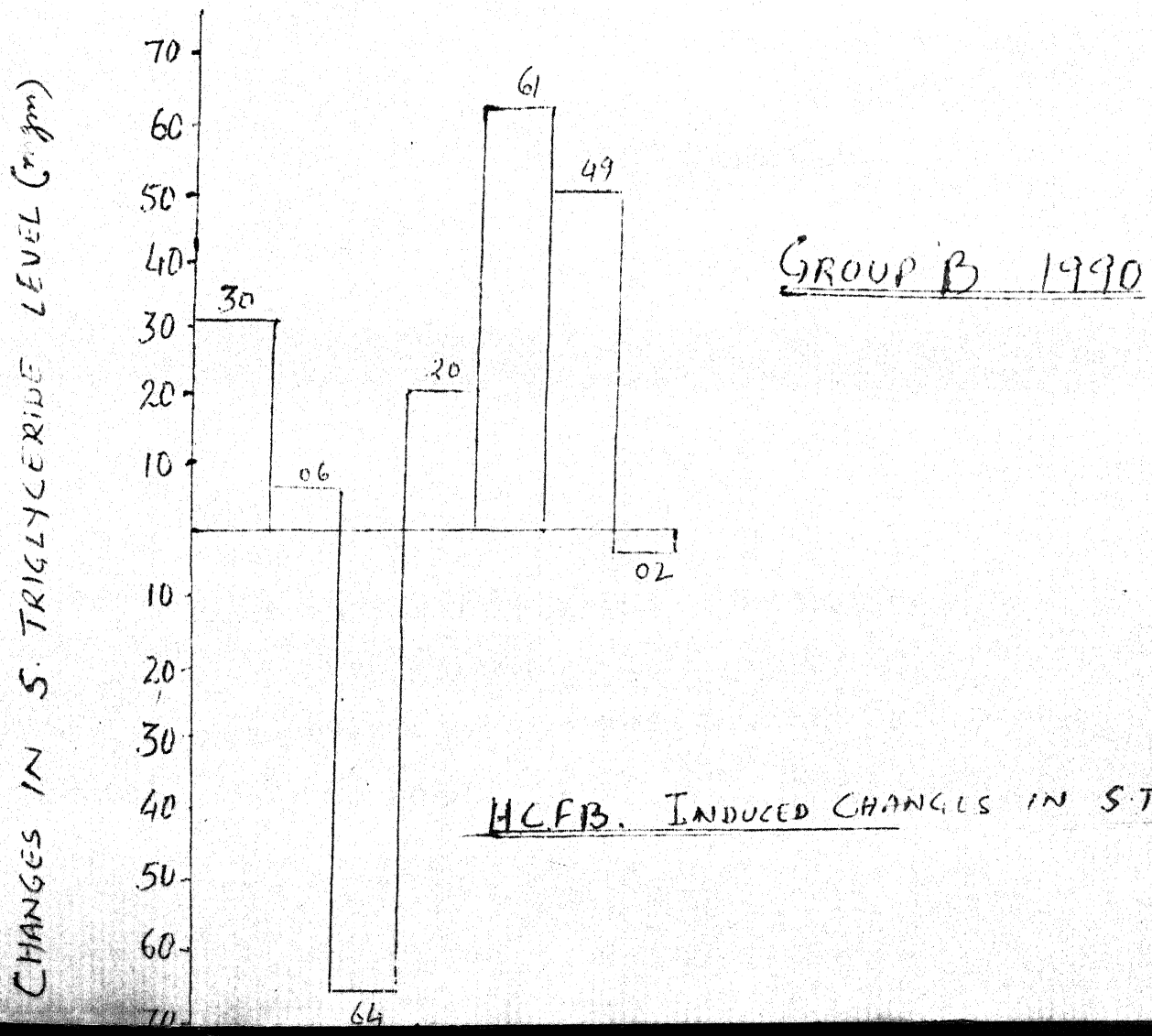
H.C.F.B. INDUCED CHANGES IN S.T.G.



HCFB. INDUCED CHANGES IN S.T.G.



H.C.F.B INDUCED CHANGES IN S.T.G.



HCFB. INDUCED CHANGES IN S.T.G.

Fig. Fig. 2, a bar diagram shows the difference in 1984 and 1990.

Thus HCFB induced changes are not reproducible after a gap of 5-6 years both in adolescents and middle aged persons.

In 1984, there was a significant rise in STC after HCFB. In 1990 also there was a significant rise but the quantum is less.

HCFB Induced Variations in STG

Group A

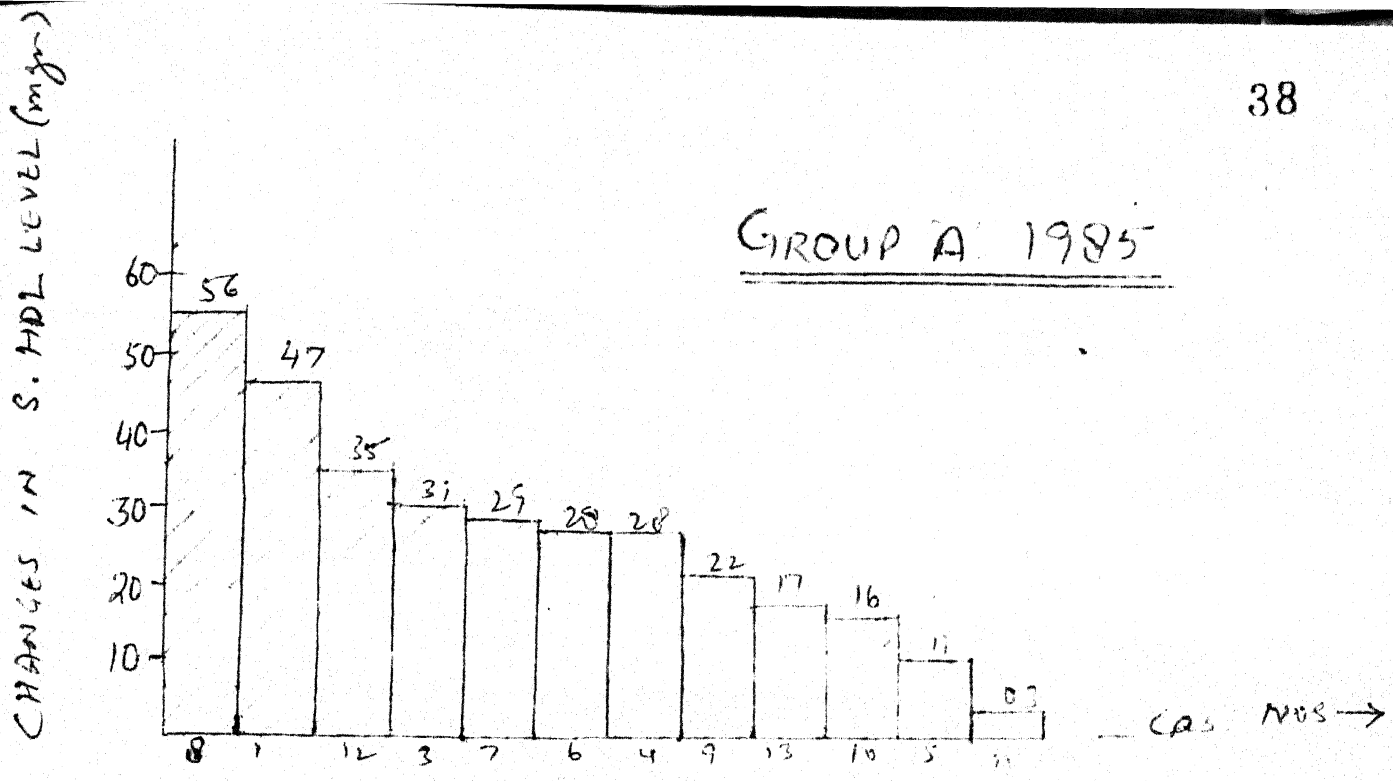
HCFB induced changes in STG in 1985 was 23.9$\pm$17.39 mg/dl. The corresponding figure for 1990 was 17.17$\pm$14.53 mg/dl. The difference was statistically significant and hence HCFB induced changes in STG in adolescents were not reproducible after a gap of 5 years ($p < 0.05$).

Fig. 3 represents the HCFB induced changes in group A in 1985 and 1990.

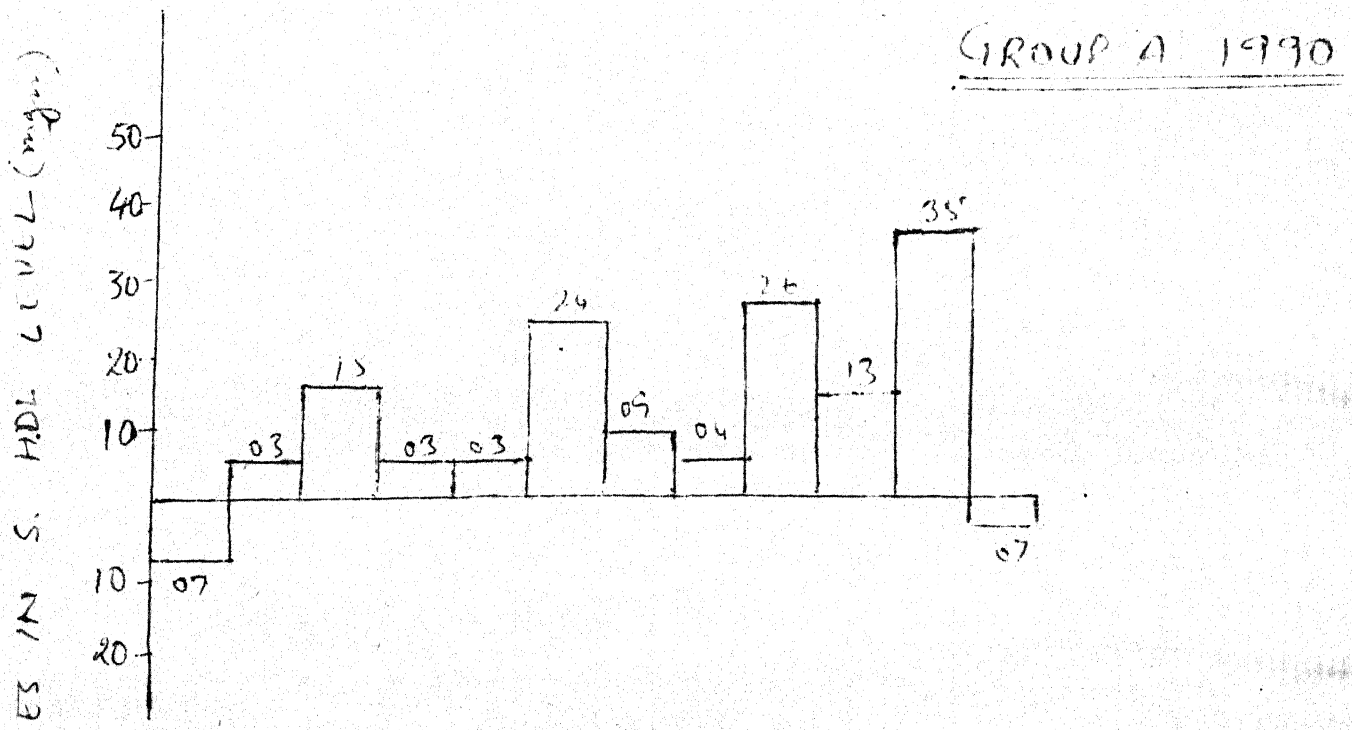
Group B

HCFB induced changes in STG were 23.71$\pm$15.66 mg/dl in 1984 and 33.14$\pm$25.39 mg/dl in 1990. The difference was statistically significant ($p < 0.05$).

HCFB induced changes in STG were not reproducible after a gap of 5 years in middle aged persons.

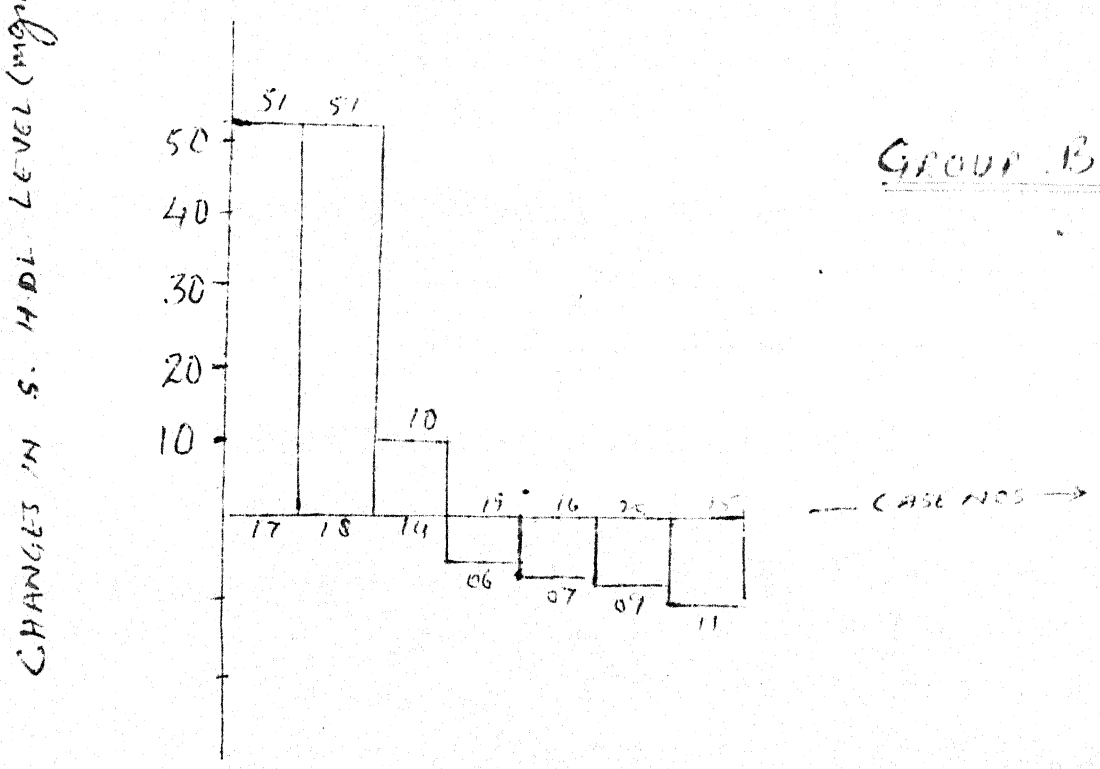


H. C. F B. INDUCED CHANGES IN S HDL



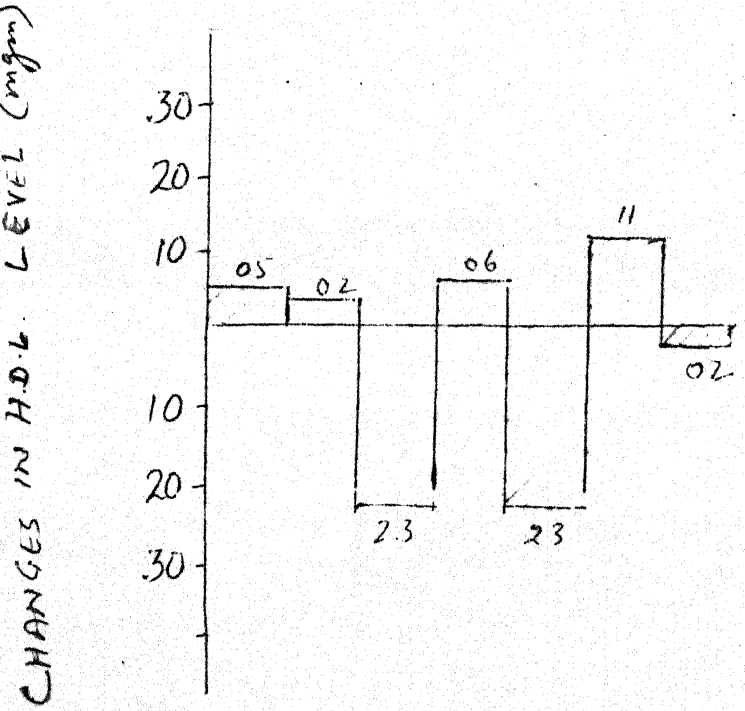
CH & B. INDUCED CHANGES IN S. HDL

GROUP B. 1989



H.C.F.B. INDUCED CHANGES IN S HDL

GROUP B 1990



H.C.F.B. INDUCED CHANGES IN S. HDL

HCFB induced changes in STG were not reproducible in adolescents and middle aged persons.

Fig. 4 is a bar diagram of HCFB induced changes in group B in 1984 and 1990.

VARIATION IN S. HDL LEVEL FOLLOWING HCFB

Group A

HCFB induced changes in HDL levels in 1985 was 26.92±14.78.mg/dl. The corresponding values for 1990 was 12.00±10.95 mg/dl. The difference at the two occasions was statistically significant($P < 0.05$).

Group B

HCFB induced changes in HDL in 1984 was 20.71 ±20.75 and corresponding mean for 1990 was 10.20±9.19 mg/dl. The difference was statistically significant ($p < 0.05$).

Fig. 6 is the graphic representation of the variations in serum HDL during the two studies.

Hence HCFB induced changes in HDL levels were not reproducible in adolescents as well as middle aged persons after a gap of 5-6 years.

Case No. 1 (Mr. Sameem)

Lipid Lipo-protein	1985			1990		
	I	II	III	I	II	III
STC	117	164	143	111	130	110
STG	115	140	98	81	79	128
HDL	37	84	36	37	40	42
LDL	57	52	87	57.8	74.2	42.4
VLDL	23.0	28.0	19.6	16.2	15.8	25.6
LDL/HDL	1.54	0.62	2.41	1.56	1.85	1.01

Changes in fasting lipid profile levels after HCFB (I-II) and after withdrawal of HCFB (I-III).

Lipid lipo-protein	1985		1990	
	I-II	I-III	I-II	I-III
STC	+47	+26	+19	-1
STG	+29	-17	- 2	+47
HDL	+47	- 1	+ 3	+ 5
LDL	- 5	+30	+16.4	-15.4
VLDL	+ 5	-4.4	-0.4	+ 9

Master Sameem aged 13 years increased from 17 kg to 35 kg in weight and his height was increased from 114 cms to 155 cms. He was a non smoker and non-alcoholic till 1990.

In 1985 his basal fasting STC was 117 mg/dl while LDL/HDL ratio was 1.54. When exposed to HCFB his STC was increased by 47 mg/dl to 164 mg/dl and LDL/HDL

ratio was 0.62, because all the cholesterol increase was contributed by HDL when HCFB was withdrawn STC was 149 mg/dl and LDL/HDL ratio was 2.41. This person routine meals imposed low risk, HCFB induced risk wasl still lower on account of major increase in HDL, but delayed effect which persisted after withdrawal of HCFB carried a comparatively higher risk but was still in low risk range.

In 1990, basal fasting STC was 111 mg/dl and LDL/HDL was 1.56 which was very well comparable to 1985 value. When exposed to HCFB for 7 days, STC was 130 mg/dl and LDL/HDL ratio was 1.85 following withdrawal of HCFB STC was 110 mg/dl and LDL/HDL was 1.01. Thus high cholesterol fat induced risk was low and remained low(delayed effect) after 7 days of withdrawal of HCFB.

Thus both in 1985 and 1990 HCFB induced risk was low.

Case No. 3 (Avdhesh)

Lipid lipo-protein	1985			1990		
	I	II	III	I	II	III
STC	156	173	147	116	154	119
STG	123	169	136	52	79	90
HDL	25	56	38	38	41	37
LDL	106.0	83.0	82.0	67.6	97.2	64.0
VLDL/	24.6	33.8	27.2	10.4	15.8	18.0
LDL/HDL	4.24	1.48	2.16	1.78	2.37	1.73

Changes in fasting lipid profile levels after HCFB (I-II) and after withdrawal of HCFB(I-III)

Lipid lipo-protein	1985		1990	
	I-II	I-III	I-II	I-III
STC	+17	- 9	+38	+ 3
STG	+46	+13	+27	+38
HDL	+31	+13	+ 3	- 1
LDL	-23	-24	+29.6	- 3.6
VLDL	+9.2	+2.6	+5.4	+ 7.6

Avdhesh aged 15 years, a student, whose height was increased from 140 to 162 cms and weight was increased from 26 to 45 kg and was non smoker and non alcoholic till 1990.

In 1985, basal fasting STC was 156 mg/dl and LDL/HDL ratio was 4.24 that was a routine meal induced moderate risk on account of low HDL level. When exposed to HCFB STC was 173 mg/dl and LDL/HDL ratio was 1.48 following 7 days withdrawal of HCFB. STC was 147 and LDL/HDL was 2.16. The HCFB induced risk was low and remained low after withdrawal of HCFB.

In 1990, STC was 116 mg/dl and LDL/HDL ratio was 1.78 following HCFB STC was 154 mg/dl and LDL/HDL ratio was 2.37 and following withdrawal STC was 119 mg/dl and LDL/HDL was 1.73. Thus routine meals and HCFB induced risk was low and persisted to be low following 7 days withdrawal of HCFB.

In this case in 1985 routine meal imposed moderate risk while in 1990 same meals imposed low risk. HCFB induced risk was low both in 1985 and 1990. The risk remained unchanged after 7 days of withdrawal of HCFB.

Case No. 4 (Mr. Santosh)

Lipid lipo-protein	1985			1990		
	I	II	III	I	II	III
STC	190	216	208	202	185	180
STG	100	108	156	175	143	158
HDL	50	78	50	34	43	53
LDL	120.0	116.0	137.0	133.0	113.4	95.4
VLDL	20.0	21.6	21.2	35.0	28.6	31.6
LDL/HDL	2.40	1.49	2.74	3.90	2.62	1.80

Changes in fasting lipid profile levels after HCFB(I-II) and after withdrawal of HCFB(I-III).

Lipid lipo-protein	1985		1990	
	I-II	I-III	I-II	I-III
STC	+26	+18	-17	-22
STG	+ 8	+ 6	-32	-17
HDL	+28	-	+ 9	+19
LDL	- 4	+17	-19.6	-37.6
VLDL	+1.6	+1.2	-6.4	-3.4

Santosh was a 16 years old student. His weight was increased from 22 to 38 kg and height from 136 to

168 cms. He was non smoker and non alcoholic till 1990.

In 1985, his basal fasting STC was 190 mg/dl and LDL/HDL was 2.4 after 7 days of HCFB feeding. STC was increased by 26 mg/dl and LDL/HDL was 1.49. After withdrawal of HCFB STC was 208 mg/dl and LDL/HDL ratio was 2.74. Thus the person with low risk to routine diet when exposed to HCFB the diet induced risk was moderate and remained moderate even after withdrawal of HCFB.

In 1990, basal fasting STC was 202 mg/dl and LDL/HDL ratio was 3.9 when HCFB was given for 7 days STC was 185 mg/dl and LDL/HDL was 2.62 after withdrawal of HCFB STC was 180 mg/dl and LDL/HDL ratio was 1.8.

Thus this case was at high risk with his routine daily meals.

HCFB induced risk was low which remained low after when tested 7 days after withdrawal of HCFB.

Thus in 1990, in this case with HCFB the risk was less compared to that with routine meals.

When the findings of 1985 and 1990 were compared it was found that after a gap of 5 years, this subject was tolerated HCFB better than in 1985.

His routine meal in 1985 imposed a low risk, but the same routine meals in 1990 was imposing high risk. The cause of this behaviour of lipid profile to routine meals was not explanable as there was practically no change in his dietary habit and life style.

Case No. 5 (Mr Mohan)

Lipid lipo-protein	1985			1990		
	I	II	III	I	II	III
STC	162	245	208	148	180	160
STG	125	150	121	125	160	125
HDL	78	89	67	35	70	45
LDL	59	126	116.8	88	78	90
VLDL	25	30	24.2	25	32	25
LDL/HDL	0.76	1.42	1.74	2.51	1.11	2.0

Changes in fasting lipid profile levels after HCFB(I-II) and after withdrawal of HCFB (I-III)

Lipid Lipo-protein	1985		1990	
	I-II	I-III	I-II	I-III
STC	+83	+46	+32	+12
STG	+25	- 4	+35	-
HDL	+11	-11	+35	+10
LDL	+67	+57.8	-10	+ 2
VLDL	+ 5	- 0.8	+ 7	-

Mohan was a a mess servant of 16 years age. His weight and height were increased from 31 to 48 kg and 129 to 160 cms respectively during last 5 years. He was consuming high fat diet.

In 1985, basal fasting STC was 162 mg/dl and LDL/HDL ratio was 0.76 when HCFB was given for 7 days STC rose by 83 mg/dl to 245 mg/dl and LDL/HDL ratio was 1.42 after 7 days of withdrawal of HCFB STC was 208 mg/dl and

LDL/HDL ratio was 1.74. That was HCFB induced risk was moderate which persisted to be moderate after 7 days of withdrawal of HCFB.

1990, the basal fasting STC was 148 mg/dl and LDL/HDL ratio was 2.51 that was diet induced risk was low. When HCFB was given for 7 days STC rose to 180 mg/dl and LDL/HDL ratio 1.11 following withdrawal STC was 160 mg/dl and LDL/HDL ratio was 2. Thus HCFB induced risk was low and remained low after 7 days of withdrawal of HCFB.

This case when tested in 1985 was consuming high fat diet when tested in 1990 he was still consuming high fat diet as he was working in doctors mess. HIS HCFB induced risk which was moderate in 1985 was found to be low in 1990.

Case No. 6 (Mr. Deep Chand)

Lipid lipo-protein	1985			1990		
	I	II	III	I	II	III
STC	106	145	106	148	170	150
STG	100	115	110	150	160	150
HDL	25	53	25	36	60	40
LDL	61	69	59	82	78	80
VLDL	20	23	22	30	32	30
LDL/HDL	2.44	1.3	2.36	2.28	1.3	2.0

Changes in fasting lipid profile levels after HCFB (I-II) and after withdrawal of HCFB(I-III).

Lipid lipo-protein	1985		1990	
	I-II	I-III	I-II	I-III
STC	+39	-	+22	+ 2
STG	+15	+10	+10	-
HDL	+28	-	+24	+ 4
LDL	+ 8	- 2	- 4	- 2
VLDL	+ 3	+ 2	+ 2	-

Deep Chand was a tempodriver in 1990, who was a mess servant when 1st study was done in 1985. He was 17 years old. His height and weight increased from 150 to 175 cms and 30 to 56 kg respectively. He was a non smoker and non alcoholic till 1990.

In 1985, basal fasting STC was 106 mg/dl while LDL/HDL ratio was 2.44. When HCFB was given STC rose by 39 mg/dl to 145 mg/dl. Main contribution was by HDL hence LDL/HDL ratio was only 1.3. After withdrawal of HCFB STC came down to previous level of 106 mg/dl but LDL/HDL ratio was 2.36. Thus HCFB induced risk was low following HCFB and after its withdrawal.

In 1990, basal fasting STC was 148 mg/dl and LDL/HDL ratio was 2.28 following HCFB for 7 days STC was 170 mg/dl that was 22 mg/dl higher and wholly contributed by HDL and hence LDL/HDL was only 1.3. After withdrawal STC returned to previous level i.e. 150 mg/dl and LDL/HDL ratio was 2.

Hence HCFB induced risk was low both in 1985 and 1990 and at both occasions it remained low following 7 days of withdrawal.

Case No. 7 (Mr. Ashok)

Lipid lipo-protein	1985			1990		
	I	II	III	I	II	III
STC	169	213	201	182	160	180
STG	138	160	157	150	150	160
HDL	57	86	79	60	63	65
LDL	84.4	95.0	90.6	92.0	67.0	83.0
VLDL	27.6	32.0	31.4	30.0	30.0	32.0
LDL/HDL	1.48	1.10	1.15	1.53	1.04	1.27

Changes in fasting lipid profile levels after HCFB(I-II) and after withdrawal of HCFB(I-III).

Lipid lipo-protein	1985		1990	
	I-II	I-III	I-II	I-III
STC	+44	+32	-22	-2
STG	+22	+19	-	+10
HDL	+29	+22	+ 3	+ 5
LDL	+10.6	+6.2	-25.0	-9.0
VLDL	+4.4	+3.8	-	+2.0

Ashok Kumar aged 18 years, a mess servant was working in Doctor's mess for last 6 years. His height and weight were increased from 145 to 168 cms and 39.5 to 57 kg respectively. He was nonsmoker and non non

alcoholic till 1990.

In 1985, basal fasting STC was 169 mg/dl and LDL/HDL was 1.48 when he was given HCFB for 7 days STC rose by 44 mg/dl to 213 mg/dl and LDL/HDL ratio was 1.1 because major contribution in STC rise was given by HDL. After withdrawal of HCFB STC was 201 mg/dl and LDL/HDL was 1.15. Thus HCFB induced risk was moderate which persisted even after withdrawal of HCFB ad delayed effect.

In 1990, basal fasting STC was 182 mg/dl and LDL/HDL ratio was 1.53 after 7 days of HCFB STC was 160 mg/dl and LDL/HDL ratio was 1.06 and after 7 days of withdrawal of HCFB STC was 190 mg/dl and LDL/HDL ratio was 1.27 that was in 1990 HCFB induced risk remained low. after HCFB and also following withdrawal.

Being a mess servant person was consuming high cholesterol fat diet. In 1985 he had shown a moderate risk to HCFB BUT in 1990, HCFB induced risk was low.

Case No. 8 (Mr. Ashok S/o Sri Chandrika Pd)

Lipid lipo-protein	1985			1990		
	I	II	III	I	II	III
STC	121	147	143	155	170	133
STG	130	138	136	102	146	114
HDL	19	75	47	43	36	33
LDL	76.0	45.0	69.0	91.6	104.8	77.2
VLDL	26.0	27.6	27.2	20.4	29.2	22.8
LDL/HDL	4.0	0.6	1.46	2.1	2.87	2.33

Changes in fasting lipid profile levels after HCFB(I-II) and after withdrawal of HCFB(I-III).

Lipid lipo-protein	1985		1990	
	I-II	I-III	I-II	I-III
STC	+26	+22	+15	-22
STG	+ 8	+ 6	+44	+12
HDL	+56	+28	- 7	-10
LDL	-31	- 7	+13.2	-14.4
VLDL	+1.6	+1.2	+8.8	+2.4

Ashok was 18 year old whose weight was increased from 28 to 56 kg and height was increased from 140 to 165 cms. He was a non smoker and non alcoholic till 1990. During last five year he was consuming low fat diet.

In 1985, his basal fasting STC was 121 mg/dl of which LDL was 76 mg/dl, VLDL - 26 mg/dl and HDL was very low at 19 mg/dl and hence LDL/HDL ratio was 4. His routine diet induced risk was moderate. After giving HCFB there was a drastic increase in HDL (56 mg/dl) while total STC was increased only by 26 mg/dl and was 147mg/dl. Hence LDL/HDL ratio was 0.6 only. Following withdrawal of HCFB STC was 143 and LDL/HDL ratio was 1.46. Thus diet induced risk with routine meals was itself carrying moderate risk, but when HCFB was given, HDL rose drastically and HCFB induced risk was thus low, which remained low as a delayed effect after withdrawal of HCFB.

In 1990, basal STC was 155 mg/dl and LDL/HDL ratio was 2.1. When HCFB was given STC was 170 mg/dl and LDL/HDL ratio was 2.87. Thus HCFB induced risk was low

and remained low when HCFB was withdrawn.

His dietary habit in last 5 years and life style was not changed. HCFB induced risk was low both in 1985 and 1990.

Case No. 9 (Mr Kailash)

Lipid lipo-protein	1985			1990		
	I	II	III	I	II	III
STC	162	167	169	171	171	194
STG	112	170	150	160	175	175
HDL	64	86	71	56	60	40
LDL	75.6	47.0	68.0	83.0	76.0	119.0
VLDL	22.4	34.0	30.0	32.0	35.0	35.0
LDL/HDL	1.18	0.55	0.95	1.48	1.27	2.90

Changes in fasting lipid provile levels after HCFB(I-II) and after withdrawal of HCFB (I-III).

Lipid lipo-protein	1985		1990	
	I-II	I-III	I-II	I-III
STC	+5	+ 7	-	+23
STG	+58	+38	+17	+15
HDL	+22	+ 7	+ 4	-16
LDL	-28.6	-7.6	- 7	+36
VLDL	+11.6	+7.6	+3.0	+3.0

Kailash, a mess servant aged 19 years male, a non smoker, non alcoholic. His weight and height were increased from 37.5 to 50 kg and from 135 cms to 166 cms in last five years. He consumed moderate fat diet.

In 1985, basal STC was 162 mg/dl and LDL/HDL ratio was 1.18. Following HCFB STC was 167 mg/dl but there was an increase of HDL by 22 mg/dl and decrease in LDL by 28.6 mg/dl so that LDL/HDL ratio was only 0.55. After 7 days of withdrawal of HCFB STC was 168 mg/dl and LDL/HDL ratio was 0.95. Thus HCFB induced risk was all time low in 1985.

In 1990, STC was 171 mg/dl and LDL/HDL was 1.48 following HCFB STC remained constant while LDL/HDL ratio was 1.27 and after withdrawal of HCFB STC rose to 194 mg/dl and LDL/HDL ratio was 2.9. Thus in 1990, HCFB induced risk was low.

Hence both in 1985 and 1990 the risk with routine meals and with HCFB was low.

Case No. 10 (Mr. Sanjai)

Lipid lipo-protein	1985			1990		
	I	II	III	I	II	III
STC	112	156	156	126	144	113
STG	146	154	150	83	85	100
HDL	37	53	72	31	44	35
LDL	45.8	72.2	54.0	78.4	83.0	58.0
VLDL	29.2	30.8	30.0	16.6	17.0	20.0
LDL/HDL	1.24	1.36	0.75	2.52	1.88	1.66

Changes in fasting lipid profile levels after HCFB(I-II) and after withdrawal of HCFB(I-III).

Lipid lipo-protein	1985		1990	
	I-II	I-III	I-II	I-III
STC	+44	+44	+18	-13
STG	+ 8	+ 4	+ 2	+17
HDL	+16	+35	+13	+ 4
LDL	+26.4	+8.2	+4.6	-20.4
VLDL	+1.6	+0.8	+0.4	+3.4

Sanjai, a 19 years old student was consuming low fat diet. His activities were moderate. He was non alcoholic, non smoker till 1990. His weight increased from 45.5 kg to 50 kg and height was increassed from 160 to 168 cms.

In 1985, basal fasting STC was 112 mg/dl and LDL/HDL ratio was 1.24, so that normal diet had low risk. When exposed to HCFB STC rose by 44 mg/dl to 156 mg/dl more contribution was by LDL and hence LDL/HDL ratio was 1.36 following withdrawal of HCFB for 7 days, STC remained at 156 mg/dl but HDL rose and LDL fell with the result that LDL/HDL ratio was 0.75 thus in 1985 for subject Sanjai HCFB induced risk was low at both occasions.

In 1990, basal fasting STC level was 126 mg/dl and LDL/HDL ratio was 2.52 following HCFB STC rose by 18 mg/dl to 144 mg/dl. Wholly contributed by HDL. LDL/HDL ratio was 1.88 following withdrawal of HCFB STC fell to 113 and fall was attributed chiefly by LDL, so that LDL/HDL ratio was 1.66 for this subject in 1990 HCFB induced risk was low at both occasions.

Thus we see that both in 1985 and 1990 HCFB induced risk remained low following feedings and following withdrawal.

Case No. 11 (Mr. Chandan)

Lipid lipo-protein	1985			1990		
	I	II	III	I	II	III
STC	165	204	165	154	143	124
STG	154	154	156	111	118	148
HDL	44	47	33	32	30	29
LDL	90.2	126.2	100.8	99.8	89.4	65.4
VLDL	30.8	30.8	31.2	22.2	23.6	29.6
LDL/HDL	2.05	2.68	3.05	3.12	3.0	2.26

Changes in fasting lipid profile levels after HCFB(I-II) and after withdrawal of HCFB(I-III).

Lipid lipo-protein	1985		1990	
	I-II	I-III	I-II6	I-III
STC	+39	-	-11	-30
STG	-	+ 2	+ 7	+37
HDL	+ 3	-11	- 2	- 3
LDL	+36	+10.6	+10.4	-34.4
VLDL	-	+0.4	+1.4	+7.4

Mr. Chandan was a 21 years old student. He did moderate exercise and was on low fat diet. His weight was increased from 50 to 54 kgs and his hieght was increased from 164 to 168 cms.

In 1985 basal fasting STC was 165 mg/dl and LDL/HDL ratio was 2.05 when HCFB was given for 7 days STC increased by 39 mg/dl to 204 mg/dl and LDL/HDL was 2.68. That was HCFB induced risk increased and was moderate.

Following withdrawal of HCFB for 7 days STC came to basal level of 165 mg/dl. Then LDL/HDL ratio remained 3.05. Thus following withdrawal of HCFB the high fat cholesterol induced risk continued to be moderate.

In 1990, basal fasting STC was 154 mg/dl and LDL/HDL ratio was 3.12 when subjected to HCFB. STC fell down to 143 mg/dl and LDL/HDL ratio was 3. Following withdrawal of HCFB STC still fell and was 124 mg/dl. The fall was mainly in LDL and hence LDL/HDL ratio was 2.26. For subject Chandan in 1990 for routine meals and HCFB induced risk was moderate but follow withdrawal risk was low.

Thus in 1985 and 1990 HCFB induced risk was moderate at both occasion risk remained moderate following withdrawal of HCFB while in 1990 following withdrawal risk was low.

Case No. 12 (Mr. Kishan)

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Lipid lipo-protein	1985			1990		
	I	II	III	I	II	III
STC	176	207	1092	182	200	190
STG	118	156	128	90	110	85
HDL	86	121	107	60	75	65
LDL	66.4	54.8	59.4	110.0	103.0	108.0
VLDL	23.6	31.2	25.6	18.0	22.0	17.0
LDL/HDL	0.77	0.45	0.55	1.73	1.37	1.66

Changes in fasting lipid lipoprotein levels after HCFB (I-II) and after withdrawal of HCFB (I-III).

Lipid lipo-protein	1985		1990	
	I-II	I-III	I-II	I-III
STC	+31	+16	+18	+ 8
STG	+38	+10	+20	- 5
HDL	+35	+21	+15	+ 5
LDL	-11.6	- 7	- 1	+ 4
VLDL	+7.6	+ 2	+ 4	- 1

Kishan was 22 years old. He was a student in 1985 and was a autorikshaw driver for the last 2 years. He was consuming moderate amount of fat . His weight was increased from 50 to 53 kgs and his height was increased from 158 to 168 cms. He was a non smoker,non alcoholic till 1990.

In 1985, basal fasting STC was 176 mg/dl and LDL/HDL ratio was 0.77 as HDL was high (86 mg/dl), when exposed to HCFB STC increased by 31 mg/dl to 207 mg/dl. The increase was wholly contributed by HDL 135 mg/dl hence LDL/HDL ratio remained 0.45 following withdrawal of HCFB STC 192 mg/dl . The increased was due to HDL. LDL/HDL ratio was 0.55.

Thus Kishan in 1985 showed moderate risk to HCFB as STC was 207 mg/dl but more than 50% cholesterol was HDL.Following withdrawal the diet induced risk was low.

In 1990, STC was 182 mg/dl and LDL/HDL ratio was 1.73 following HCFB for 7 days, STC increased by 18 mg/dl

to 200 mg/dl and was wholly contributed by HDL and hence LDL/HDL ratio was 1.37 follow HCFB withdrawal STC was 190 mg/dl with LDL/HDL ratio was 1.66.

In 1990, the HCFB induced risk was moderate but was low following withdrawal.

Comparing 1985 and 1990 it was observed that at both occasions HCFB induced moderate risk and risk was low following withdrawal.

Case No. 13 (Mr. Daulat)

Lipid lipo-protein	1985			1990		
	I	II	III	I	II	III
STC	245	246	157	260	160	156
STG	150	180	162	160	150	150
HDL	42.0	59.0	56.0	40.0	66.0	66.6
LDL	173.0	151.0	68.6	188.0	64.0	59.4
VLDL	30.0	36.0	32.4	32.0	30.0	30.0
LDL/HDL	4.12	2.55	1.23	4.70	0.97	0.89

Changes in fasting lipid profile levels after HCFB(I-II) and after withdrawal of HCFB (I-III).

Lipid lipo-protein	1985		1990	
	I-II	I-III	I-II	I-III
STC	+ 1	-88	-100	-104
STG	+30	+12	-10	-10
HDL	+17	+14	+26	+26.6
LDL	-22	-104.4	-124	-128.6
VLDL	+ 6	+2.4	- 2	- 2

Daulat was a 22½ years old mess servant. His weight increased from 50 to 55 kg and his height has increased from 158 to 165 cms. He was non alcoholic and non smokers till 1990.

In 1985, basal fasting STC was 245 and LDL/HDL was 4.72 that was with routine diet, he was at high risk. When HCFB was given, STC remained constant but there was a fall in LDL (22 mg/dl) and an increase in HDL(17 mg/dl) so that LDL/HDL ratio was 2.55. Following withdrawal, STC was 157 mg/dl and LDL/HDL ratio was 1.23. The fall in STC was wholly due to fall in LDL.

In 1990, STC was 260 mg/dl and LDL/HDL ratio was 4.12. LDL level was higher (188 mg/dl) following HCFB STC was 160 mg/dl and fall was totally due to fall in LDL 124 mg/dl. LDL/HDL ratio was 2.9. Following withdrawal STC was 156 mg/dl and LDL/HDL ratio was 0.89.

Thus in 1990 routine meals induced risk was high. HCFB induced risk was low and persistent low following withdrawal of HCFB.

In 1985 and 1990 this case showed high level of STC. LDL was the chief contributor at both occasions. With HCFB the risk decreased. Decrease was at earlier period in 1990 while comparatively less in 1985, in both the years following withdrawal of diet induced risk was low.

The behaviour of this subject was abnormal for reason not known at both occasions basal fasting LDL level was high which came down on feeding HCFB.

Case No. 14 (Mr. Munshi Lal)

Lipid lipo-protein	1984			1990		
	I	II	III	I	II	III
STC	143	162	205	158	220	156
STG	85	100	80	222	158	159
HDL	62	72	66	72	49	47
LDL	64.0	70.0	123.0	41.6	139.4	77.2
VLDL	17.0	20.0	16.0	44.4	31.6	31.8
LDL|HDL	1.03	0.97	1.86	0.58	2.80	1.64

Changes in fasting lipid profile levels after HCFB (I-II) and after withdrawal of HCFB(I-III).

Lipid lipo-protein	1984		1990	
	I-II	I-III	I-II	I-III
STC	+19	+62	+62	- 2
STG	+15	- 5	-64	-63
HDL	+10	+ 4	-23	-25
LDL	+ 6	+59	+97.8	+31.8
VLDL	+ 3	- 1	-12.8	-12.6

The subject Munshi Lal aged 52 years was a chronic alcoholic consuming 150-180 ml of Desi alcohol since last 15-16 years. He was a bidi smoker(20 bidi/day) for the last 20 years. He was an Ex. army personnel. He has put up 4 kg weight in last 6 years.

In 1984, STC was 143 mg/dl and LDL/HDL ratio was 1.03. When exposed to HCFB for 7 days STC was 162 mg/dl and LDL/HDL ratio was 0.97. After withdrawal of HCFB for

7 days STC was 205 mg/dl and LDL/HDL ratio was 1.86. That was routine diet had low risk. HCFB induced was low and remained low following withdrawal of HCFB.

1990, STC was 158 mg/dl and LDL/HDL ratio was 0.58 when exposed to HCFB STC was 220 mg/dl and LDL/HDL ratio was 2.84. After withdrawal of HCFB, THE STC was 156 mg/dl and LDL/HDL ratio was 1.64. This showed that in 1990 HCFB induced risk was moderate. It was low when seen 7 days after withdrawal. of HCFB.

Comparing 1984 to 1990 it was observed that this subject's risk to HCFB has increased in 1990. This habit of chronic bidi smoking, chronic alcoholism and aging factor all must have contributed in this change.

Case No. 15 (Kishore Kumar)

Lipid lipo-protein	1984			1990		
	I	II	III	I	II	III
STC	136	149	164	130	128	160
STG	85	140	80	120	150	150
HDL	61	50	58	27	25	27
LDL	58	71	90	79	73	103
VLDL	17	28	16	24	30	30
LDL/HDL	0.95	1.42	1.55	3.0	3.0	3.8

changes in fasting lipid profile levels after HCFB(I-II) and after withdrawal of HCFB (I-III).

Lipid lipo-protein	1984		1990	
	I-II	I-III	I-II	I-III
STC	+13	+28	- 2	+30
STG	+55	- 5	+30	+30
HDL	-11	- 3	- 2	-
LDL	+13	+32	- 6	+24
VLDL	+11	- 1	+ 6	+ 6

The subject Kishore Kumar aged 50 years was an alcoholic, consuming 100-120 ml daily since last 10-12 years. He was a bidi smoker for the same duration. He has put 2 kg weight in last 6 years. He was on low fat diet. His weight was within normal limit for this height. He has In 1984, his basal fasting STC was 136 mg/dl and LDL/HDL ratio was 0.95 when exposed to HCFB the STC was 149 mg/dl. LDL/HDL ratio was 1.42 and after withdrawal of HCFB STC was 164 mg/dl and LDL/HDL ratio was 1.55. Thus it was observed that HCFB induced risk was low.

In 1990, basal fasting STC was 130 mg/dl and LDL/HDL ratio was 3. When exposed to HCFB STC was 128 mg/dl and LDL/HDL ratio was 3 and after withdrawal of HCFB, the STC was 160 mg/dl and LDL/HDL ratio was 3.8 that is STC remained well within normal limit at all three occasions but LDL/HDL ratio was above 3 at all the three occasions.

Thus in 1984 diet induced risk was low while in 1990 diet induced risk was moderate.

This change can be attributed to smoking, alcohol consumption and ageing factors.

Case No. 16 (Mr. Chandra Bahadur)

Lipid lipo-protein	1984			1990		
	I	II	III	I	II	III
STC	195	150	164	150	203	174
STG	91	100	160	86	147	184
HDL	68	61	58	58	35	35
LDL	109.0	69.0	74.0	74.8	138.6	102.2
VLDL	18.2	20.0	32.0	17.2	29.4	36.8
LDL/HDL	1.6	1.13	1.27	1.29	3.8	3.0

Changes in fasting lipid profile levels after HCFB (I-II) and after withdrawal of HCFB (I-III).

Lipid lipo-protein	1984		1990	
	I-II	I-III	I-II	I-III
STC	-45	-31	+53	+24
STG	+ 8	+68	+61	+98
HDL	- 7	-10	-23	-23
LDL	-40	-35	+63.4	+27.4
VLDL	+1.8	+13.8	+12.2	+19.6

Chandra Bahadur aged 48 years, a non alcoholic has put up 6 kg weight in last 6 years. He was an over weight. He was bidi smoker, smoking 15-16 bidi/day for the last 25 years.

In 1984, his basal fasting STC was 195 mg/dl and LDL/HDL ratio was 1.6. When exposed to HCFB for 7 days STC was 150 mg/dl and LDL/HDL ratio was 1.13. After withdrawal of HCFB STC was 164 mg/dl and LDL/HDL ratio was 1.27. Thus routine meal induced risk was low and HCFB induced risk was also low.

In 1990, basal fasting STC was 150 mg/dl and LDL/HDL ratio was 1.29. HCFB induced STC was 203 mg/dl and LDL/HDL ratio was 3.8 when HCFB was withdrawn for 7 days STC was 174 mg/dl and LDL/HDL ratio was 3. Thus it was observed that although after 6 years routine meals induced risk was low but HCFB induced risk was moderate and remained moderate after withdrawal of HCFB.

The subject after 6 years had a greater diet induced risk which could be attributed to over weight ageing factor and smoking habit.

Case No. 17 (Mr. Vijai Singh)

Lipid lipo protein	1984			1990		
	I	II	III	I	II	III
STC	169	273	190	170	159	187
STG	85	60	120	50	48	68
HDL	62	113	66	38	43	44
LDL	90	148	100	122	106.4	129
VLDL	17	12	24	10	9.6	14.0
LDL/HDL	1.45	1.31	1.51	3.2	2.47	3.0

Changes in fasting lipid profile levels after HCFB(I-II) and after withdrawal of HCFB (I-III).

Lipid Lipo-protein	1984		1990	
	I-II	I-III	I-II	I-III
STC	+104	+21	-11	+17
STG	-25	+35	- 2	+18
HDL	+51	+ 4	+ 5	+ 6
LDL	+58	+10	-15.6	+ 7
VLDL	- 5	+ 7	-0.4	+4.0

Vijai Singh aged 46 years had normal weight for his height. He was non smoker and non alcoholic. There has been no change in dietary intake and life style in last 6 years.

In 1984, basal fasting STC level was 169 mg/dl and LDL/HDL ratio was 1.45. Thus he was at low risk to routine meals. In response to HCFB STC was 273 mg/dl and LDL/HDL ratio was 1.31 that is risk to HCFB was moderate. Following withdrawal of HCFB STC was 191 mg/dl, LDL/HDL ratio was 1.5. Thus HCFB induced risk was moderate while it was again low following withdrawal of HCFB.

In 1990, STC was 170 mg/dl and LDL/HDL ratio was 3.2 that was after these 6 years the routine meals induced risk was moderate. The risk was low followed by HCFB for 7 days as STC was 159 mg/dl and LDL/HDL ratio was 2.27.

After withdrawal of HCFB STC was 187 mg/dl and LDL/HDL ratio was 3. Thus we see that after gap of 6 years his risk to routine meals has increased and HCFB

gave a delayed risk which persisted after withdrawal of HCFB.

This change could be a contributed to age factor only as other parameters were unchanged.

CASE No. 18 (Mr. Raghubir)

Lipid lipo-protein	1984			1990		
	I	II	III	I	II	III
STC	130	247	171	109	124	151
STG	71	100	184	86	92	121
HDL	60	111	56	31	33	36
LDL	56	116	78	60.8	72.6	90.8
VLDL	14.2	20.0	36.8	17.2	18.4	24.2
LDL/HDL	0.93	1.05	1.39	1.96	2.2	2.52

Changes in fasting lipid profile levels after HCFB(I-II) and after withdrawal of HCFB(I-III).

Lipid lipo-protein	1984		1990	
	I-II	I-III	I-II	I-III
STC	+117	+41	+15	+42
STG	+29	+113	+ 6	+35
HDL	+51	- 4	+ 2	+ 5
LDL	+60	+22	+11.8	+30
VLDL	+5.8	+22.6	+1.2	+7.0

Mr. Raghubir aged about 51 year, was a bidi smoker for the last 20 years. He was non alcoholic doing moderate exercise. There was no change in his life style or dietary habit.

In 1984, STC was 130 mg/dl and LDL/HDL ratio was 0.93, when exposed to HCFB for 7 days STC was 247 mg/dl and LDL/HDL ratio was 1.05. After withdrawal of HCFB for 7 days STC was 171 mg/dl and LDL/HDL ratio was 1.39 thus in 1984 he was at low risk with routine meals. HCFB induced risk was moderate. After withdrawal, HCFB induced risk was low.

In 1990, STC was 109 mg/dl and LDL/HDL was 1.96 after HCFB STC was 124 mg/dl and LDL/HDL was 2.2 and after withdrawal of HCFB, STC was 151 mg/dl and LDL/HDL was 2.52.

Thus we see that this case in 1984 HCFB induced risk was moderate while in 1990 compared to that with routine meals HCFB induced risk was low.

This subject has given abnormal behaviour that in diet induced risk was low in 1990 compared to that of 1984 inspite that person was bidi smoker.

CASE No. 19 (Mr. Sarman)

Lipid lipo-protein	1984			1990		
	I	II	III	I	II	III
STC	146	162	164	164	176	185
STG	142	120	184	88	137	166
HDL	65	59	60	29	35	33
LDL	53	79	64	117.4	113.6	118.8
VLDL	28.4	24.0	40.0	17.6	27.4	33.2
LDL/HDL	0.82	1.33	1.06	4.00	3.24	3.60

Changes in fasting lipid profile levels after HCFB(I-II) and after withdrawal of HCFB(I-III).

Lipid lipo-protein	1984		1990	
	I-II	I-III	I-II	I-III
STC	+16	+18	+12	+21
STG	-22	+42	+49	+79
HDL	- 6	- 5	+ 6	+ 4
LDL	+26	+11	-3.8	+1.4
VLDL	-4.4	+12	+10.2	+15.6

A 46 years old man whose weight remained constant during last 6 years. His dietary intake and life style also remained unchanged. He was bidi smoker for the last 16 years smoking 15-20 bidi daily. He was non alcoholic.

In 1984, basal fasting STC was 146 mg/dl and LDL/HDL ratio was 0.82 so that case was low rish with normal diet. When exposed to high HCFB for 7 days STC rose to 183 mg/dl and LDL/HDL ratio was 1.33 and 7 days after withdrawal of HCFB STC was 164 mg/dl and LDL/HDL ratio was 1.06. That is to say that this subject remained at low risk during high cholesterol feeding and also during the period of 7 days of withdrawal.

In 1990, basal fasting STC was 164 mg/dl but LDL/HDL ratio was 4. The case was thus having moderate risk with routine diet. With 7 days of HCFB STC was 176 mg/dl and LDL/HDL ratio was 3.24 that is moderate risk and after 7 days of withdrawal STC was 185 mg/dl and LDL/HDL ratio was 3.6.

In 1984 the case was low risk with normal diet and remained at low risk with HCFB and following its withdrawal. This case in 1990 became moderate risk to normal diet and remained moderate risk to HCFB and following withdrawal. This could be on account of ageing factor and smoking as other factors remained unchanged.

CASE No. 20 (Mr. Kashi Ram)

Lipid lipo-protein	1984			1990		
	I	II	III	I	II	III
STC	195	247	195	198	220	188
STG	100	112	160	68	88	109
HDL	81	72	63	33	44	32
LDL	94	153	100	151.4	158.4	134.2
VLDL	20.0	22.4	32.0	13.6	17.6	21.8
LDL/HDL	1.16	2.13	1.58	4.39	3.6	4.19

Changes in fasting lipid profile after HCFB (I-II) and after its withdrawal (I-III).

Lipid lipo-protein	1984		1990	
	I-II	I-III	I-II	I-III
STC	+52	+ 3	+22	-10
STG	+12	+60	+20	+41
HDL	- 9	-18	+11	- 1
LDL	+59	+ 6	+ 7	-17.2
VLDL	+2.4	+12	+ 4	+8.2

Mr. Kashi Ram aged 52 years, has put 4 kg weight in last 6 years. He was non alcoholic. He smoked 10-11 bidi/day for the last 20 years. His feeding habit and

life style have not changed.

In 1984, basal fasting STC was 195 mg/dl and LDL/HDL ratio was 1.16. That was low risk to routine feeds. Following 7 days HCFB the STC was 247 mg/dl and LDL/HDL ratio was 2.13 and 7 days after withdrawal of HCFB STC was 198 mg/dl and LDL/HDL ratio was 1.58 that was HCFB exposed the person to moderate risk following the risk was low.

In 1990, STC was 198 mg/dl and LDL/HDL ratio was 4.59 that is person turned to moerate risk to his normal feeds. In response to HCFB STC was 220 mg/dl and LDL/HDL ratio was 3.6 that is high risk. Following withdrawal of HCFB STC was 188 mg/dl and LDL/HDL ratio was 4.99 that is moderate risk.

In 1984 the subject had moderate risk to HCFB. In 1990 HCFB induced risk was high. It was moderate following withdrawal of HCFB.

After 6 years this person developed greater risk to HCFB. His risk was also increased to his routine meals.

The possible explanation for this change can be ageing factor and smoking as other parameter remained unchanged.

DIET INDUCED RISK

The routine diet induced risk and HCFB induced risk was calculated in individual cases on the basis of arbitrary scale, details of which is given in material and method.

TABLE 11 : Routine diet incuded risk in group A and B.

Risk	Group A		Group B	
	1985	1990	1984	1990
Low	9(75%)	9(75%)	7(100%)	3(43%)
Moderate	2(17%)	1(8%)	-	4(57%)
High	1(8%)	2(17%)	-	-

Table 11 shows that routine diet induced risk in adolescents(group A) was low in most (75%) of the cases in both 1985 and 1990.

In old age d persons routine diet imposed a low risk in 1984 in all the cases but with the same diet the risk was increased in 57% cases in 1990.

TABLE 12 : HCFB induced risk in group A and B.

Risk	Group A		Group B	
	1985	1990	1984	1990
Low	6(50%)	10(83.3%)	4(57%)	1(14%)
Moderate	6(50%)	2(16.7%)	3(43%)	5(72%)
High	-	-	-	1(14%)

HCFB induced risk in group A was low in 50% cases and was moderate in 50% in 1985. The risk was low in 83.3% cases and moderate in 16.7% cases only in 1990.

Hence it is observed that adolescents group were tolerating HCFB better than in 1985.

In subjects of group B HCFB induced risk has increased in 1990 in comparison to 1984.

ROUTINE DIET INDUCED RISK ON INDIVIDUAL LEVEL

Group A

This was worked out on basis of basal fasting serum lipid values. Of the 12 subjects, 7(58%) cases No. 1,5,6,7,9,10,12) were such that routine diet was imposing a low risk both in 1985 and 1990.

In one subject (8%)(No. 13) the risk was high both in 1985 and 1990.

In two(17%) subjects(No. 3,8) routine diet induced risk was moderate in 1985 but it was low in 1990.

In two subjects(No. 4, 11)(17%) routine diet induced risk was low in 1985 but increased in 1990.

Thus routine diet induced risk in 1990 when compared to that of 1985, it was found that in 65% cases it has remained same, in 17.5% cases it reduced while in 17.5% cases risk has increased.

Group B

Of the 7 subjects studied, routine meal induced risk was low in all the subjects in 1984. In 1990, 3(43%) cases(No. 14,16,18) showed low risk, while 4(57%) cases (No. 15,17,19) 20) showed an increase to moderate risk to routine meals.

In general in the adolescent subjects routine meal induced risk has not increased. In middle aged aubjects, routine meal induced risk has increased in most of the subjects.

HCFB INDUCED RISK

Group A

Of the 12 subjects studied, 6(50%) cases(No. 1,3, 6,8,9,10) HCFB induced risk was low in 1985 and was found low also in 1990.

In 2(17%) cases (No. 11,12) HCFB induced risk was moderate in 1985 as well as in 1990 also.

In 4(33%) subjects(No.4,5,7,13) HCFB induced risk was moderate in 1985 while it was low in 1990.

Thus in 1990 in 67% cases HCFB induced risk has remained unchanged while in 33% cases that was reduced.

The HCFB induced risk has not increased in any of the adolescent subject.

GROUP B

Of the 7 subjects examined, 4(57%) cases No. 14,15 16, 19) in whom HCFB induced risk was low in 1984 but in 1990 the HCFB induced risk was moderate.

In one case (No. 20) HCFB induced risk which was moderate in 1984 has become high in 1990. In another one subject (No. 17) HCFBiinduced risk has remained the same during the two studies.

In one subject (No. 18) HCFB induced risk decreased in 1990.

Thus in 72% cases HCFB induced risk has increased in 1990 as compared to that of 1984. In 14% cases (1 case) it reduced and in 14%(1 case) cases it remained unchanged.

DISCUSSION

D I S C U S S I O N

The present study was under taken on 12 healthy subjects of age group 13-23 years(Group A) and 7 healthy subjects of age group 45-55 years (Group B).

The method of study and protocol were exactly the same as were in the previous studies of 1984 and 1985. The time gap in the two studies was 5 years for group A and 6 years for group B.

The study was aimed to test the reproducibility of basal fasting lipid profile and HCFB induced lipi profiles. It was also aimed to study the reproducibility of variations in lipid profile induced by HCFB. The diet induced risk was also studied.

REPRODUCIBILITY OF BASAL FASTING LIPID PROFILE

Serum Total Cholesterol (STC)

Fasting STC level prior to test meal was reproducible in both group A and group B. The differences observed were statistically insignificant(P γ0.05).

Serum Triglyceride (STG)

Fasting STC levels prior to test meal, were strongly reproducible in group A while in group B the reproducibility was comparatively weak(p γ0.05).

HIGH Density Lipoprotein(HDL)

Fasting HDL levels were reproducible in group A while in group B the changes observed were highly

significant (p $\angle$0.05) and hence were not reproducible (Table 7 and 8).

Serum Total Cholesterol(STC)

It is generally accepted that over night fasting serum cholesterol level in an individual fluctuate within a narrow range and this pattern is mentained for at least 5 years.

The present study confirmed the generally accepted concept.

Serum Triglyceride

The present study showed that fasting STG level in an individual fluctuates within a narrow range and hence is reproducible this is in accordance with generally accepted belief.

Serum HDL

In group A, serum HDL levels were reproducible. In group B they were not reproducible. There was a steep decline in mean HDL level from 65.5$\pm$7.32 in 1984 to 41.14 $\pm$17.14 mg%. This variation in group B could be because of within subject variations.

REPRODUCIBILITY OF FASTING LIPID PROFILE FOLLOWING HCFB FOR 7 DAYS

Serum Total Cholesterol

Fasting STC levels after 7 days of HCFB were

not reproducible in both group A and B. The differences observed (Table 3 and 4) were significant ($p < 0.05$).

The response of serum cholesterol to HCFB is erratic and unpredictable, an increase in STC level was observed in most of the subjects but some of them showed a fall in STC level.

Beynen and Katan (1985) studied the effect of cessation of egg consumption on STC levels. They concluded that the STC levels were only weakly reproducible.

The present study shows that STC levels are not reproducible. This difference in observation could be because the amount of fat and cholesterol content in test meal of present study was much higher than the test meal of above mentioned study (egg consumption was withdrawn and each subject was consuming on average 1.6 egg daily).

STG

The difference observed in HCFB induced STG level were statistically significant $p < 0.05$ and hence were not reproducible in both group A and group B.

The study of Cohen, Noakes and Benade (1988) concluded that STG levels following high fat diet (upto 80 gm fat) increased steadily for first seven hours following which STG levels showed erratic response. For how long these responses persisted was not mentioned. This study also concluded that with a diet of 80 gms fat the STG level increased in proportion to amount of fat consumed beyond 80 gm that is at 120 gm s. This pattern

was last. In this study reproducibility was studied after 21 days with 80 gms fat, a 19% variation in serum triglyceride level was found.

In our study HCFB was given for 7 days hence total fat consumption was very high and secondly STG levels were seen after 24 hours of last test meal.

Serum HDL

HCFB induced HDL level were not reproducible in both group A and group B.($p < 0.05$). This finding confirms the previous findings of Vega and Grandy that serum HDL and serum cholesterol levels are not consistent with dietary intake.

REPRODUCIBILITY OF HCFB INDUCED CHANGES IN LIPID PROFILE

STC

Both in group A and B the differences observed in HCFB induced STC levels (I-II) are highly significant ($p < 0.05$). Fig. 1 and 2 represent HCFB induced changes in bar diagram. The pattern indicates their non reproducibility.

In study of Beynen and Katan (1985) the subjects were studied at an interval of 6 years. Each subject was consuming an average of 1.6 egg/day for last 15 days. Variation in STC to cessation of egg consumption were observed and compared they concluded that variations were only weakly reproducible.

The present study shows that variations are non reproducible after a gap of 5-6 years.

This difference could be because the test diet used in this study was very large. Thus the present study observation coincide with the general till date observation that diet induced changes in STC were erratic.

STG

In both the group A and B the difference in HCFB induced changes in STG were statistically strongly significant and hence were not reproducible (Table 9 and 10). Similar studies on the subject are not available.

The observations of Cohen, Noakes and Benade (1988) suggested that changes in serum triglyceride levels increased consistently when test meal consisted of 80 gms fat or less, at more than 80 gms fat the changes were erratic. Similarly the changes were uniform for 1st seven day following test meal. Later on they were erratic. Reproducibility tested after 21 days show a variation of 19%.

The present study test meal consisted of fat about 80 gms. Our study confirms the findings of Cohen and Benade, (1985).

HDL

Difference in HCFB induced changes in HDL levels were strongly statistically significant and hence were not reproducible (Table 9 and 10).

This was true for both group A and B. Work on the subject of reproducibility after 6 years has not been done and hence in present study findings cannot be compared.

Elaborate studies on larger sample size are required to establish the above statement.

ROUTINE DIET INDUCED RISK

Group A

Routine diet induced risk was low in 75% cases in 1985. The risk remained low in 75% cases in 1990 also.

In only one case the risk to routine diet has increased. This could be a matter of chance, otherwise in all cases routine diet induced risk has remained low both in 1985 and 1990.

Group B

There was a definite increase in routine diet induced risk in middle aged subjects(increase was observed in 4 cases, 57% cases).

This could be that at an age in between 45-55 years body ability to tolerate routine cholesterol fat load decreases.

A study on larger group can establish more accurately the age limits at which these changes occur.

Thus we can advise on the basis of our study that person in middle age group should keep restriction on their diet.

HCFB INDUCED RISK

Group A

In 1985, 50% subjects had a higher risk to HCFB while it was low in 50% cases in 1990. 83% cases showed low risk and only 2 cases were with higher risk.

This could be that the test diet provided was very high. In 1985 the mean age of group A was 13±2.8 years and hence they could not tolerate this high test meal but the same subjects now with mean age of 18±2.86 years were able to tolerate the same test diet.

Group B

There was a definite increase in HCFB induced risk in group B (Table 12). Thus there is some turning point in age at which HCFB induced risk increase. The findings are in accordance to the accepted rule that with advancing age tolerance to diet decreases.

SUMMARY AND CONCLUSION

SUMMARY AND CONCLUSION

The present study was carried out in 19 healthy volunteers. The selection of the subjects was done from the subjects who were studied in previous studies conducted in our department in 1984 and 1985. The selection was made on the basis of their availability and being declared healthy as per set criteria.

The selected subjects were divided into two group - Group A and Group B. Group A consisted of 12 healthy subjects of age group 13 to 23 years and group B consisted of 7 healthy middle aged subjects of age group 45-55 years.

The protocol and method of study in the previous studies and in the present study were essentially the same.

The selected subjects were subjected to an over night fast of 14 hours. They were not allowed to take any thing else except water prior to withdrawal of blood samples.

Basal fasting blood sample(I) was taken after an over night fast. Subjects were provided with test meal (High cholesterol fat breakfast) in place of their routine meals for 7 days. On day 8th, fasting blood sample (II) was taken. Test meal was withdrawn and subjects was allowed to take his routine breakfast. On 15th day fasting blood sample (III) was taken.

High cholesterol fat breakfast consisted of :

a. 4 slices of average size.

b. 25 gm Amul posturized butter.

c. 2 egg omellet prepared in 20 gm vanaspati ghee.

d. 250 ml of whole fat buffalo milk(sweetened).

e. 1 gm of crystalline cholesterol.

The blood samples were analysed for serum total cholesterol, serum triglycerides, serum HDL,. From these values LDL, VLDL and LDL/HDL ratio were calculated.

The aims of the study were to test the reproducibility of HCFB induced lipid profile, and work out diet induced risk.

Reproducibility of Basal fasting Lipid Profile

Basal fasting serum cholesterol levels, serum triglyceride levels were reproducible in both the groups A and B, while HDL was reproducible in group A only and not reproducible in middle aged subjects.

Reproducibility of HCFB induced serum lipid profile

It was seen that following HCFB for 7 days STC, STG and HDL none of these were reproducible.

Reproducibility of HCFB Induced Variations in Lipid Profile

The variations (difference of I and II) induced by HCFB, were not reproducible in cases of serum cholesterol, serum triglyceride and serum HDL.

Routine Diet Induced Risk

Routine diet induced risk was low in group A i.e. 13-23 years age group during both the studies. In group B i.e. in middle aged subjects the routine diet induced risk was increased in most of the cases in 1990.

HCFB Induced Risk

HCFB induced risk is low in younger subjects. In group B i.e. middle aged subjects, HCFB induced risk has increased in 1990.

The following conclusions were drawn from the present study.

1. Basal fasting serum cholesterol levels are reproducible in younger as well as middle aged persons for atleast 5 years.
2. Basal fasting serum triglycerides levels were reproducible in younger as well as middle aged persons for atleast 5 years.
3. Basal fasting HDL level were reproducible in younger subjects but were non reproducible in middle aged subjects after 5 years.

   In middle aged subjects, HDL level had fallen after 5 years of study gap.
4. High cholesterol fat breakfast induced cholesterol level, serum triglyceride level and serum HDL level were not reproducible and showed gross inconsistency in post prandial behaviour and hence cholesterol tolerance test is not feasible and practicable

at this stage.

5. HCFB induced variations in serum cholesterol, serum triglyceride level and serum HDL level were not reproducible i.e. the variations are erratic and are not predictable.

6. Routine diet induced low risk in young persons.

7. HCFB induced risk is low in adolescents.

8. HCFB induced risk is higher in middle aged persons.

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MASTER CHART

Group 'A'

MASTER - CHART

Sl. No.	Name and Father's name	Address	Age/Sex (yr)	Wt. (kg)	Ht. (cms.)	Physical activity	Dietary habit	Occupation	Smoking	Alcoholism	Appox. daily fat intake(g)
1.	Sameem S/o B.Khan	Yellow Qrs. Med. Coll. Campus, JHS.	13/M	35	155	Moderate	N.V.	Student	NS	NA	35
2.	Ved Prakash S/o V. Singh	-do-	14½/M	46	160	"	Veg.+ egg	"	NS	NA	35
3.	Avdhesh S/o Vijai Singh	-do-	15/M	45	162	"	"	"	NS	NA	35
4.	Santosh S/o Late M. Singh	-do-	16/M	38	168	"	"	"	NS	NA	27
5.	Mohan S/o Shukhlal	Kochhabhanwar Jhansi	16/M	48	160	"	"	Mess Servant	NS	NA	39.5
6.	Deep Chand S/o Raja Ram	Talpura, JHS	17/M	56	175	"	"	Tempo Driver	NS	NA	21
7.	Ashok S/o Har Prasad	Pichhore, JHS	18/M	57	168	"	NV	Mess Servant	NS	NA	45
8.	Ashok S/o C. Prasad	Yellow Qrs. Med.Coll. JHS	18/M	56	165	"	NV	Student	NS	NA	22
9.	Kailash S/o Har Charan	Bara Bazar, Jhansi.	19/M	50	166	"	NV	Mess servant	NS	NA	31
10.	Sanjai S/o Sarman Lal	Yellow Qrs. Med.Coll. JHS.	19/M	50	168	"	NV	Student	NS	NA	26
11.	Chandan S/o SK Singh	-do-	21/M	54	168	"	Veg.	Student	NS	NA	29
12.	Kishan S/o Ram Charan	Behind Med.Coll. Near Kumhar JHS.	22/M	53	168	"	NV	Auto	NS	NA	49
13.	Daulat S/o Har Prasad.	Pichhore, JHS.	22½/M	55	165	"	NV	Mess servant	NS	NA	38

Group 'B'

14.	Munshi Lal Ward Boy.	Ward VIII, MediColl.JHS.	52/M	58	161	Moderate	Veg.+ egg	Ward boy	S	A	HF
15.	Kishore Kumar	-do-	58/M	55	160	Heavy	NV	Sweeper	S	A	LF
16.	C.Bahadur	-do-	48/M	64	159	"	NV	Chokidar	S	NA	LF
17.	Vijai Singh	-do- Lib.	46/M	49	160	Moderate	Veg.+ egg	Atten- dant	NS	NA	LF
18.	Rabhubir	-do-	51/M	54	161	"	NV	Peon	S	NA	LF
19.	Sarman H/o Raj Kumari Aya	-do-	46/M	64	163	"	Veg.+ egg	Home work	S	NA	LF
20.	Kashi Ram	-do-	52/M	62	160	"	"	Chowki- dar	S	NA	LF
21.	Banwari	-do-	51/M	58	168	Heavy	"	Sweeper	S	A	LF

M = Male, Mod. Moderate, NV = Non-vegetarian, NS = Non-smoker, S = smoker.
NA= Non-alcoholic, A = Alcoholic, HF = High fat, LF = Low fat.

MASTER CHART

Sl. No.	Year	STC			STG			HDL		
		I	II	III	I	II	III	I	II	III
1.					Group 'A'					
1.	1985	177	164	143	115	140	98	37	84	36
	1990	111	130	110	81	79	128	37	40	42
2.	1985	130	177	138	108	116	110	19	55	33
	1990	142	-	-	125	-	-	46	-	-
3.	1985	156	173	147	123	169	136	25	56	38
	1990	116	154	119	52	79	90	38	41	37
4.	1985	190	216	208	100	108	156	50	78	50
	1990	202	185	180	175	143	158	34	43	53
5.	1985	162	245	208	125	150	121	78	89	67
	1990	148	180	160	125	160	125	35	70	45
6.	1985	106	145	106	100	115	110	25	53	25
	1990	148	170	150	150	160	150	36	60	40
7.	1985	169	213	201	138	160	157	57	86	79
	1990	182	160	180	150	150	160	60	63	65
8.	1985	121	147	143	130	138	136	19	75	47
	1990	155	170	133	102	146	114	43	36	33
9.	1985	162	167	169	112	170	150	64	46	70
	1990	171	171	194	160	175	175	56	60	40
10.	1985	112	156	156	146	154	150	37	53	72
	1990	126	144	113	83	85	100	31	44	35
11.	1985	165	204	165	154	154	156	44	47	33
	1990	154	143	124	111	118	148	32	30	29
12.	1985	176	207	192	118	156	128	86	121	107
	1990	182	200	190	90	110	85	60	75	65
13.	1985	245	246	157	150	180	162	42	59	56
	1990	260	160	156	160	150	150	40	66	66.6

.....cont.

Group 'B'

14.	1984	143	162	205	85	100	80	62	72	66
	1990	158	220	156	222	158	159	72	49	47
15.	1984	136	149	164	85	140	80	61	50	58
	1990	130	128	160	120	150	150	27	25	27
16.	1984	195	150	164	91	100	160	68	61	58
	1990	150	203	174	86	147	184	58	35	35
17.	1984	169	273	190	85	60	120	62	113	66
	1990	170	159	187	50	48	68	38	43	44
18.	1984	130	247	171	71	100	184	60	111	56
	1990	109	124	151	186	92	121	31	33	36
19.	1984	146	162	164	142	120	184	65	59	60
	1990	164	176	185	88	137	166	29	35	33
20.	1984	195	247	195	100	112	160	81	72	63
	1990	198	220	188	68	88	109	33	44	32
21.	1984	156	182	190	71	100	160	84	100	78
	1990	118	-	-	70	-	-	66	-	-

Group 'A'

MASTER - CHART

Sl. No.	Year	LDL			VLDL			LDL/HDL		
		I	II	III	I	II	III	I	II	III
1.	1985	57.0	52.0	87.0	23.0	28.0	19.6	1.54	0.62	2.41
	1990	57.0	74.2	42.4	16.2	15.8	25.6	1.56	1.85	1.01
2.	1985	89.0	100.0	84.0	21.6	23.2	22.0	4.68	1.82	2.54
	1990	71.0	-	-	25.0	-	-	1.54	-	-
3.	1985	106.0	83.0	82.0	24.6	33.8	27.2	4.24	1.48	2.16
	1990	67.6	97.2	64.0	10.4	15.4	15.8	1.78	2.37	1.73
4.	1985	120.0	116.0	137.0	20.0	21.6	21.2	2.40	1.49	2.74
	1990	133.0	113.4	95.4	35.0	28.6	31.6	3.90	2.62	1.80
5.	1985	59.0	126.0	116.8	25.0	30.0	24.2	0.76	1.42	1.74
	1990	88.0	78.0	90.0	25.0	32.0	25.0	2.51	1.11	2.00
6.	1985	51.0	69.0	59.0	20.0	23.0	22.0	2.44	1.30	2.36
	1990	82.0	78.0	80.0	30.0	32.0	30.0	2.28	1.30	2.00
7.	1985	84.4	95.0	90.6	27.6	32.0	31.4	1.48	1.10	1.15
	1990	92.0	67.0	83.0	30.0	30.0	32.0	1.53	1.04	1.27
8.	1985	76.0	45.0	69.0	26.0	27.6	27.2	4.00	0.60	1.46
	1990	91.6	104.8	77.2	20.4	29.2	22.8	2.10	2.87	2.33
9.	1985	75.0	47.0	68.0	22.4	34.0	30.0	1.18	0.55	0.95
	1990	83.0	76.0	119.0	32.0	35.0	35.0	1.48	1.27	2.90
10.	1985	45.8	72.2	54.0	29.2	30.8	30.0	1.24	1.36	0.75
	1990	78.4	83.0	58.0	16.6	17.0	20.0	2.52	1.88	1.66
11.	1985	90.2	126.2	100.8	30.8	30.8	31.2	2.05	2.68	3.05
	1990	99.8	89.4	65.4	22.2	23.6	29.6	3.12	2.98	2.26
12.	1985	66.4	54.8	59.4	23.6	31.2	25.6	0.77	0.45	0.55
	1990	104.0	103.0	108.0	18.0	22.0	17.0	1.73	1.37	1.66
13.	1985	173.0	151.0	68.6	30.0	36.0	32.4	4.12	2.55	1.23
	1990	188.0	64.0	59.4	32.0	30.0	30.0	4.70	0.97	0.89

contd...

Group 'B'

14.	1984	64.0	70.0	123.0	17.0	20.0	16.0	1.03	0.97	1.86
	1990	41.0	139.4	77.2	44.4	31.6	31.8	0.58	2.80	1.64
15.	1984	58.0	71.0	90.0	17.0	28.0	16.0	0.95	1.42	1.55
	1990	79.0	73.0	103.0	24.0	30.0	30.0	3.00	3.00	3.80
16.	1984	109.0	69.0	74.0	18.2	20.0	32.0	1.60	1.13	1.27
	1990	74.8	138.6	102.2	17.2	29.4	36.8	1.29	3.80	3.00
17.	1984	90.0	148.0	100.0	17.0	20.0	24.0	1.45	1.31	1.51
	1990	122.0	106.4	129.4	10.0	9.6	14.0	3.20	2.47	3.00
18.	1984	56.0	116.0	78.0	14.2	20.0	36.8	0.93	1.05	1.39
	1990	60.8	72.6	90.8	17.2	18.4	24.2	1.96	2.20	2.52
19.	1984	53.0	79.0	64.0	28.4	24.0	40.0	0.82	1.33	1.06
	1990	117.4	113.6	118.8	17.6	27.4	33.2	4.0	3.24	3.60
20.	1984	94.0	153.0	100.0	20.0	22.4	32.0	1.16	2.13	1.58
	1990	151.4	158.4	134.2	13.6	17.6	21.8	4.39	3.60	4.19
21.	1984	58.0	62.0	80.0	14.2	20.0	32.0	0.69	-	-
	1990	38.0	-	-	14.0	-	-	0.57	-	-

MASTER CHART

Lipid lipoprotein profile (mean ± S.D. mg/dl).

Lipid lipo protein	Year	I	II	III
		Group 'A'		
STC	1985	156.75±39.30	190.25±36.15	166.25±31.10
	1990	162.90±41.10	163.90±19.64	150.75±30.70
	C.S.D.	39.45	31.46	31.00
STG	1985	125.90±18.25	149.50±21.45	134.20±21.70
	1990	119.90±39.20	129.60±34.14	131.90±29.54
	C.S.D.	30.03	29.68	25.37
HDL	1985	47.00±21.00	73.90±21.30	56.75±23.50
	1990	42.00±10.70	52.30±14.90	45.90±13.30
	C.S.D.	16.53	21.10	19.46
		Group 'B'		
STC	1984	159.14±27.25	201.57±52.59	179.57±17.87
	1990	150.74±23.65	175.70±40.53	171.60±15.79
	C.S.D.	27.00	47.00	16.70
STG	1984	94.14±22.80	106.50±23.30	138.30±45.20
	1990	105.00±52.80	117.14±41.43	136.70±39.80
	C.S.D.	14.76	33.38	40.90
HDL	1984	65.60±7.32	76.85±25.00	61.00±4.04
	1990	41.14±17.14	37.57±7.99	36.25±6.85
	C.S.D.	17.90	27.12	13.94

C.S.D. = Combined Standard Deviation of both year.